



THE UNIVERSITY LIBRARY

PROTECTION OF AUTHOR'S COPYRIGHT

This copy has been supplied by the Library of the University of Otago on the understanding that the following conditions will be observed:

1. To comply with s56 of the Copyright Act 1994 [NZ], this thesis copy must only be used for the purposes of research or private study.
2. The author's permission must be obtained before any material in the thesis is reproduced, unless such reproduction falls within the fair dealing guidelines of the Copyright Act 1994. Due acknowledgement must be made to the author in any citation.
3. No further copies may be made without the permission of the Librarian of the University of Otago.

The effects of dietary carbohydrate on muscle glycogen metabolism and performance in an 80 minute rugby simulation

Glenn A. Kearney

A thesis submitted in partial fulfillment
of the requirements for the degree of

Master of Science

at the University of Otago, Dunedin,
New Zealand.

2001

Abstract

Rugby union forward play is characterised by intense, intermittent periods of exercise requiring high levels of aerobic fitness and substantial anaerobic power. The effects of a high and low carbohydrate diet for the three days preceding a simulation trial were studied in 10 male rugby forwards (23 ± 4 yr, 189 ± 7 cm, 96 ± 9 kg). A randomised, cross-over protocol involving two 80 min rugby simulation trials, separated by one week, was used. Subjects consumed either a high carbohydrate (HCHO) diet (>8 g.kg⁻¹) or a low carbohydrate (LCHO) diet (<4 g.kg⁻¹). Vastus lateralis biopsies were taken before and after each simulation trial. Resting muscle glycogen concentration was higher following the HCHO treatment than LCHO treatment (102 ± 28 , 59 ± 17 mmol.kg⁻¹ ww; resp-, $p=0.002$) indicating that the dietary prescription was adhered to. The HCHO dietary treatment resulted in greater glycogen utilisation than the LCHO dietary treatment (54 ± 13 , 33 ± 15 mmol.kg⁻¹ ww; resp-, $p=0.02$). Performance in the simulation was determined by measuring the ability of the subjects to perform high intensity, rugby specific tasks (e.g., mauling, scrummaging, and sprinting). Each individual was assessed against his own personal best, and each task was also given a weighting depending on the contribution it has to match play. A resultant performance score (TWS) was calculated for each lap of the simulation. The difference in the TWS from the first-half to the second-half between the two treatments (HCHO-LCHO) was 4.0 units (or 4.5%), the interaction effect between treatment, and game halves, narrowly failed to attain statistical significance ($p=0.08$; effect size = 0.7). This result may have relevant

practical implications, especially considering the simulation was found to be less intense (based on HR and BLa measurements) than match play. In conclusion, the rugby simulation resulted in a large amount of glycogen utilisation, and despite the limitations of the performance instrument, and the small sample size, it appears that CHO intake in the days leading up to a match may influence the onset of fatigue in rugby forwards.

Acknowledgements

Thanks to all those who guided, encouraged, and inspired this piece of work. A special thanks to *Markus Deutsch* and the *PE school tech team* who were instrumental in the design of the simulation, it was a pleasure to work with you Markus and I look forward to more collaboration in the future. Also, thank you to my supervisors *Dr Nancy Rehrer*, and *Assoc. Prof Christine Thomson* who were extremely supportive during the writing up of this study. *Jim*, thank you for your patience during my struggle to balance work and research commitments, you are an inspirational HOD to us all. Thank you to *Dr Dave Gerrard* for his expertise and time committed to the collection of the biopsies. *Ken*, I can't thank you enough for your encouragement and feedback during the write up, your support was so important to me.

This study would not have been possible without the financial support of *Sport Science New Zealand*, *Otago Research Grants*, and *Nutra-life (NZ) Ltd*. Product was kindly donated by the following companies: *Heinz-Wattie Ltd*, *Effem foods*, *Sanitarium*, and *Nutra-life (NZ) Ltd*.

Contents

Abstract	ii
Acknowledgements	iv
Contents	v
List of Tables	vii
List of Figures	viii
List of Abbreviations	ix
1 Introduction	1
2 Literature Review	3
Background.....	3
Carbohydrate intervention studies.....	7
Physiology of rugby union forward play.....	10
Player characteristics.....	14
Dietary Intakes.....	16
Fatigue in rugby forward play.....	16
Summary.....	19
3 Methods	20
Study design.....	20
Subject characteristics.....	22
Anthropometry.....	22
Maximal aerobic capacity ($\dot{V}O_2$ max) test.....	22
Dietary interventions.....	23
Pre-simulation meal.....	23
Simulation.....	24
Simulation development.....	24

Simulation design.....	24
Familiarisation	26
Performance and physiological load measurement.....	26
Dietary analysis.....	28
Muscle biopsies.....	28
Muscle glycogen analysis.....	29
Statistical analysis.....	30
4 Results	31
Subject characteristics	31
Dietary treatments.....	31
Simulation characteristics.....	33
Activity patterns.....	33
Work: Rest ratios	34
Heart rate.....	35
Blood lactate.....	36
Muscle glycogen	37
Simulation performance.....	38
5 Discussion	40
Key findings.....	40
Simulation characteristics.....	41
Dietary treatments.....	45
Muscle glycogen	46
Simulation performance.....	47
6 Conclusion	51
References	53
Appendices	59
Appendix A Participant Information sheets.....	60
Appendix B Consent Form	65
Appendix C Diet Record booklet.....	67
Appendix D Treatment diets	71
Appendix E Individual TWS results.....	76

List of Tables

Table 2.1	Anthropometric data on rugby forwards	15
Table 4.1	Subject Characteristics, (n=10).....	31
Table 4.2	Dietary treatment composition, (n=10).....	32
Table 4.3	Mean (SD) muscle glycogen concentration in biopsy samples following HCHO and LCHO treatments with samples collected prior to (pre) and after (post) the simulation.....	37
Table 4.4	Mean (SD) TWS for first-half (laps 1-3) and second half (laps 5-7) for the HCHO, and LCHO treatment	39

List of Figures

Figure 3.1	Overview of study design	21
Figure 3.2	Simulation circuit (one lap) schematic. Units are in meters except those following the 'stand' commands, which are seconds.....	25
Figure 4.1	Relative time spent performing defined activities during match play (club-premier) (Deutsch, 2000) and the simulation.....	33
Figure 4.2	Relative time spent exercising at each W:R ratio during the simulation and match (club-premier) play (Deutsch, 2000).....	34
Figure 4.3	Relative time spent by forwards within each HR zone for the 80 min simulation (HCHO and LCHO treatments) compared to match (club-premier) play (Deutsch, 2000)	35
Figure 4.4	Mean (SD) blood lactate concentrations (mmol.l ⁻¹) during the simulation following HCHO and LCHO dietary treatments, n=10.....	36
Figure 4.5	Box and whisker plot of muscle glycogen concentration (Glucosyl units) for HCHO and LCHO dietary condition pre- and post-simulation.....	38
Figure 4.6	Difference (%) between treatments (HCHO - LCHO) in simulation performance (TWS) with associated 95% confidence intervals	39

List of Abbreviations

CHO	Carbohydrate
LCHO	Low carbohydrate dietary treatment (<4 g.kg ⁻¹)
HCHO	High carbohydrate dietary treatment (>8 g.kg ⁻¹)
W:R ratio	Work to rest ratio(s)
TWS	Total weighted score
MSS	Multiple sprint sports
vs	versus
HR	Heart rate
ww	wet weight
dw	dry weight

1 Introduction

Numerous studies have examined the nutritional requirements and dietary manipulations for optimal endurance exercise performance. Aside from water, the principal nutrient manipulated has been carbohydrate (CHO). Over fifty years ago Christensen and Hansen (1939) first demonstrated that pre-exercise CHO intake can influence the performance of continuous ($>70\% \dot{V}O_{2\max}$) endurance exercise. This formative work demonstrated that endurance capacity is impaired when individuals consume a CHO restricted diet prior to exercise. Subsequent research extended the understanding of the performance-enhancing role of pre-exercise diet. An extreme regimen incorporating exhaustive exercise, and a period of restricted CHO intake followed by consumption of large quantities of dietary CHO resulted in an increase (or 'supercompensation'), of muscle and liver glycogen stores prior to exercise. Such a regimen has been shown to prolong submaximal ($75\% \dot{V}O_{2\max}$) exercise time to exhaustion compared with individuals who ingested their habitual or a low CHO diet (Bergstrom, et al., 1967; Bergstrom and Hultman, 1966).

Since this landmark research a more moderate approach has demonstrated that glycogen supercompensation does not require the athlete to exercise to exhaustion or restrict CHO intake prior to loading. Therefore it is now widely accepted that three days of increased CHO intake of $\sim 8\text{-}10 \text{ g CHO}\cdot\text{kg}^{-1}$ bodyweight ($\text{g}\cdot\text{kg}^{-1}$) combined with tapering training, will supercompensate glycogen content in those muscles frequently active in training (Sherman, et al., 1981). The majority of

performance related research examining the effects of manipulating pre-exercise glycogen stores has concentrated on performance during prolonged, continuous running or cycling. The balance of evidence suggests that a CHO loading regime has the potential to postpone time to fatigue by up to ~20%, and improve performance by ~2 to 3% in continuous endurance exercise that lasts for a period greater than 90 minutes.

Recent studies have begun to examine the role of diet and pre-exercise muscle glycogen content on performance in sports which are played over a broad range of exercise intensities in a given game. Such work is essential if we are to determine optimal dietary regimens specific to these types of sports. Sports such as football (soccer), rugby (union and league), field hockey, ice hockey, and basketball are examples of this type of activity, and involve intermittent, highly intensive bouts of exercise, interspersed with periods of active and passive recovery. These sports can be collectively termed multiple sprint sports.

Rugby forward play can be considered an archetypal example of a multiple sprint sport. Indeed, compared with other sports it places greater demands on players to exhibit power, and resilience to high velocity impacts. Because of the unique mix of physical demands and lack of research examining the nutritional challenges facing these athletes, it is essential that we improve our current level of understanding. Therefore, the purpose of this study was to perform an acute dietary intervention targeting pre-exercise glycogen stores and subsequently examine the effects on muscle glycogen utilisation and performance in an 80-min rugby simulation.

2 Literature Review

Background

The relatively small number of studies examining the requirements of rugby and other multiple sprint sports (MSS) partially results from the inherent practical difficulties associated with conducting field studies. Until a deeper understanding of the physiological demands of MSS is obtained it will remain difficult to be certain that dietary advice pertinent to endurance athletes does indeed apply to participants in these activities. However, several important studies have recently tackled this challenging research area. These are reviewed below.

The question of what the optimal pre-competition diet is for athletes engaging in MSS has been elegantly studied with a group of elite Swedish ice hockey players (Åkermark, et al., 1996). Thirty-two players were randomly allocated to two groups: those consuming a carbohydrate rich diet ~ 57% of total energy (TE) from CHO, which provided 8.4 g.kg⁻¹ (CHO group). The control group consumed a mixed diet ~ 42% TE from CHO, which provided 6.4 g.kg⁻¹.

Two consecutive games were selected (referred to as Games 1 and 2), with subjects consuming the dietary regimens described above during the three days between the games. All participants took part in the team training sessions (two in total) undertaken between games 1 and 2. Lunches and dinners were prepared and served to all players in

the same restaurant during the test week, with players eating breakfast individually following written instructions. All extra food intakes were carefully registered. Biopsies (from the vastus lateralis) were taken three times: after Game 1, before Game 2, and after Game 2. A sub sample (n=14) was also studied using time-motion analysis during Game 2 to profile their physical activity patterns on ice.

Muscle glycogen, measured after players had consumed their assigned diets and before Game 2, was 99 mmol.kg⁻¹ ww in the CHO group compared to 81 mmol.kg⁻¹ ww in the controls (p<0.05). Net depletion (i.e after Game 2) in the CHO group was 55 mmol.kg⁻¹ ww compared to 35 mmol.kg⁻¹ ww in the controls (p<0.05). Furthermore, the CHO group had a 45% higher glycogen resynthesis than the control group. The results also demonstrated that in regular ice hockey games, muscle glycogen levels in the vastus lateralis muscle could reach low values (as low as 29 mmol.kg⁻¹ ww). The CHO group had significantly more shifts on ice, longer time on ice (playing time), and a greater skating distance compared to the control group (30% longer, i.e. 4969 m compared to 3461 m). These differences were most evident at the end of the game (Period 3). During this period, skated distance decreased by 14% in the control group in Period 3 compared to Period 1, whereas it increased 11% in the CHO group.

This study was tightly controlled, in particular the dietary regimens, which were predominately administered and recorded within a monitored team restaurant. Performance during this study was interpreted from the distance and speed skated. These variables provide a valuable description of the physical demands during ice hockey but how well they actually reflect a player's influence upon a game is debatable. In addition, these variables were measured by time-motion analysis using direct observation. For a game as fast as ice hockey this represents a challenging and potentially flawed data

collection process. Post-match video analysis of players would provide a more precise and reliable method for determining performance variables. Also, while ice hockey is a classic example of a high intensity intermittent sport, there are distinct differences between its physical demands, and those of rugby (Green and Bishop, 1976). The mode of movement between the two sports is different (skating vs running). Ice hockey also requires a smaller contribution from the upper body, and total playing time and the intensity during the game differ between the two codes (Åkermarck, et al., 1996; Deutsch, 2000; Green and Bishop, 1976; McLean, 1992). These differences must be taken into account when interpreting the relevance of these results to rugby.

More recently Balsom, Wood, et al (1999) employed a randomised, cross-over design protocol to study six male football (soccer) players in a simulated match environment. The players were subjected to an exercise and dietary regimen (either high CHO, ~65% TE from CHO, or low CHO, ~30% TE from CHO) designed to manipulate muscle glycogen concentration. This protocol resulted in pre-game muscle glycogen concentrations following the high CHO dietary treatment that were significantly higher than following the low CHO dietary regimen ($396 \pm 78 \text{ mmol.kg}^{-1} \text{ dw}$ vs $287 \pm 85 \text{ mmol.kg}^{-1} \text{ dw}$, respectively; $p < 0.05$). Post-game muscle biopsies were not obtained but movement analysis (using direct observation) showed that players performed significantly more (~33%) high intensity activities in the game played following the high CHO dietary regimen than in the game played following the low CHO diet. Also, the time spent actively pressuring the player with the ball was also significantly greater (~50%) in the game played following the high CHO diet than in the game played following the low CHO diet. Despite the small sample size, significant performance differences were observed between the two dietary regimens, although the authors provided no information concerning the subjects' usual diet.

Determination of muscle glycogen utilisation during actual rugby match play has been estimated only once. Jardine, Wiggins, et al (1988) performed a physiological examination of South African club rugby players. This included the sampling of muscle tissue from a mixture of forwards and backs. Players were divided into two groups. A control (non-loaded) group (n=8) who consumed their usual diets, and an experimental (CHO-loaded) group (n=7) who consumed a high carbohydrate (70% TE) diet for three days preceding each biopsy. A 'pre-match' biopsy was taken on the morning of a Saturday on which no match was scheduled, while a 'post-match' sample was obtained immediately after a rugby match. Muscle glycogen utilisation was significantly ($P<0.05$) greater during the match in the CHO-loaded group ($76 \pm 30 \text{ mmol.kg}^{-1} \text{ ww}$) than in the non-loaded group ($43 \pm 7 \text{ mmol.kg}^{-1} \text{ ww}$). This study also demonstrated that rugby played at club level did not cause severe muscle glycogen depletion. Unfortunately performance was not measured, therefore, although muscle glycogen content did not reach critically low levels, it cannot be assumed that performance was not influenced by either dietary regimen. In addition, the inclusion of a combination of forwards and backs could confound the results since the energetic demands of these positions are vastly different (Deutsch, 2000; McLean, 1992).

The three studies reviewed above represent the most applied research on the effects of CHO manipulation on muscle glycogen metabolism and performance in MSS that has been published in recent years. Fortunately, other methodological approaches can also assist our search for answers.

Carbohydrate intervention studies

Researchers principally use three approaches to overcome the difficulties of determining performance, metabolic load, and treatment effects during MSS. They either assess performance in isolated laboratory based tests, in simulated matches, or in real matches. Each of these methodologies has fundamental strengths and weaknesses as research tools. Match analysis represents the 'gold standard' in research and the ultimate testing ground for nutritional interventions. Unfortunately, match play also provides a restrictive testing environment therefore limited applied research has been carried out during matches. Thus, the majority of studies examining MSS have been conducted in a laboratory setting, or in simulated matches. Research employing these design elements is beginning to unravel the complex interaction of the energy systems, and how they provide adequate energy to fuel dynamic metabolic demands. However, a major limitation of laboratory based research is in the validity of the performance measure: how well do changes in an isolated physical or cognitive task translate into on-field performance? Although characteristics such as concentration, reaction time, or the ability to recover between repeated sprints may be important features in a game, one must be cautious when extrapolating performance in an isolated test to performance in a match. Although no simulated match will ever completely mimic the demands of an actual match, if it is designed with attention to detail, it should represent the closest one can get to actually testing during a real match. Reviewed below are several studies that have employed these approaches to test various acute nutritional interventions in MSS.

Jenkins, Palmer, et al (1993) tested a group of 14 moderately trained young males. Each subject completed two identical 'maximal interval tests' (MIT₁ and MIT₂) over a 10 day experimental period. Each MIT

involved five 60 sec all-out periods of cycling against a standardised body weight adjusted resistance, with each period separated by five min of passive recovery. All subjects consumed a moderate CHO diet (55% TE CHO) for three days preceding MIT₁ and were then randomly assigned to a high CHO (83%), moderate CHO (58%), or low CHO (12%) diet for the three days separating MIT₁ and MIT₂. The principal finding of this study was that a high (83%) and moderate (58%) intake of dietary CHO could at least sustain the capacity for repeated all-out exercise, when compared to a low (12%) CHO intake.

Nicholas (1997) recently used an intermittent, high-intensity, shuttle run test protocol (I-HI protocol) to investigate whether running capacity could be restored within 22 hr following consumption of a diet containing additional energy, in the form of either CHO, or fat and protein. This line of research has important implications for a tournament situation where players are required to perform optimally on successive days or with a short recovery period. The principal finding was that a normal diet supplemented with carbohydrate to the order of 10 g.kg⁻¹ improved endurance capacity during the I-HI protocol after a recovery of 22 hr. An equivalent return in exercise performance did not occur when subjects consumed an isocaloric diet containing their normal amount of CHO plus additional fat and protein during the same recovery period. These studies demonstrate that athletes participating in prolonged, intermittent high-intensity exercise may benefit from an elevated CHO intake above their normal intake, particularly if recovery time is limited.

Several investigations have attempted to measure changes in performance following acute ingestion of CHO beverages pre-game and at half-time. Leatt and Jacobs (1989) investigated the hypothesis that glucose polymer ingestion at appropriate intervals before and during a soccer match would decrease the net utilisation of muscle glycogen

during the game. It was postulated that this strategy might therefore delay the onset of fatigue induced by glycogen depletion. This investigation involved 10 male soccer players divided into two groups. Five players on the experimental team ingested 0.5 L of a 7% glucose polymer solution 10 min before the game and at half-time, while the five control team players ingested equal volumes of placebo at the same time. Biopsy samples obtained from the vastus lateralis before and after the game revealed a significantly greater decrease (i.e. greater utilisation, $p < 0.01$) in muscle glycogen in the subjects who ingested the placebo than in those who drank the glucose drink. The supplemented group finished the game with a 31% greater muscle glycogen concentration than the placebo group. Unfortunately, this field study lacked pre-intervention dietary control or a performance measure to provide the conclusive evidence necessary to develop sound nutritional recommendations.

Muckle (1973) investigated the effects of glucose ingestion on both individual and team performance in 20 soccer matches. In 10 of those games, players were allowed to drink a glucose solution, while in the other 10 games they consumed a placebo. With glucose ingestion the team scored more goals and did not concede as many, especially in the second half, in comparison with the matches in which the team consumed the placebo. Conversely, players who did not ingest glucose showed a 20-50% reduction in the number of ball contacts and involvement in play during the final 30 min of the games. Also Kirkendall, Foster, et al (1988) reported that a highly concentrated glucose polymer solution (25%) ingested immediately before a soccer match and at half-time resulted in more "running distance at speed" being covered during the second half (30%) than when no carbohydrate was consumed. It is important to recognise that the performance instrument described above provides at best, a 'blunt' measurement of performance in a MSS.

Unfortunately these intervention studies represent only acute dietary manipulation and have failed to report the influence of the diet and training load during the days before the specific intervention. While a direct ergogenic effect may be responsible for the aforementioned 'performance' benefits, it could also be argued that acute carbohydrate drink supplementation may in fact only be compensating for a sub-optimal diet and/or inadequate exercise taper before competition. This was demonstrated by Flynn, Costill, et al (1987) who observed that none of the three carbohydrate-containing beverages consumed by his subjects during a 2 hr cycling test was superior to water for athletes who had undergone glycogen loading in the two days prior to the test. Since most studies have used fasted athletes without preliminary glycogen loading, initial stores of glycogen in muscle and liver are unlikely to always be optimal in those studies. This may explain the observed benefits of CHO consumption during exercise. Future investigations should control for, and report on, dietary intake and exercise undertaken 48 hours before data collection.

Physiology of rugby union forward play

The physiological demands of rugby have received relatively little attention by the scientific community, and when pioneering studies were undertaken the demands have proved extremely difficult to isolate and quantify. A principal reason for this is the complexity and variation of movement patterns. Other factors such as the physical nature of the game, referee's influence, relative ability and tactics of the opposition, and the prevailing environmental conditions (i.e. playing surface, wind, rain, temperature, humidity etc) also contribute to create an extremely challenging and variable data collection environment.

In order to create an accurate simulated match environment (or an isolated lab test) to objectively test nutritional interventions, it is first essential to have a detailed understanding of the metabolic load of the MSS under investigation. Several techniques have been used to estimate the physiological demands of rugby union. These include time-motion analyses (Deutsch, et al., 1996; Docherty, et al., 1988; McLean, 1992; Treadwell, 1988), blood lactate (BLa) and heart rate recordings (HR), (Deutsch, 2000; Deutsch, et al., 1996; McLean, 1992) and measurement of pre-, and post-game muscle glycogen concentrations (Jardine, et al., 1988).

The initial estimations of workload in rugby union were undertaken in the 1970's and 80's with basic time-motion analysis of players the preferred data collection method (Morton, 1978; Reid and Williams, 1974). Because of the crude methodological techniques used, and the rapid evolution of the game, the data obtained from these formative studies is now of limited scientific value. However, these studies did provide the framework for subsequent, more detailed work.

McLean (1992) conducted an in-depth analysis (time-motion analysis, BLa measurements) of the 1989-90 Five Nations Championship. Players were assumed to be working when the ball was in play, and otherwise in a state of rest. Activities performed by the players were categorised as either low intensity (standing, walking, and jogging) or high intensity (running with elongated stride, sprinting, and non-running, intense activities). The average total playing time – when the ball was in play - was 29 min, representing 36% of the match duration. The average duration of a passage of play was 19 sec. A key contribution from this study was the assertion that the physiological demand on players is not only contingent on the running speed and duration, but also the density (work to rest ratios) of the physical work performed. For sports involving a range of running velocities, movement

modalities, and various static and dynamic activities, the work rate and in particular work to rest ratios (W:R ratios) of various positions provide a more descriptive data set than just measures of speed and distance alone. The W:R ratios which occurred most frequently were 1:1 to 1:1.9, with 37% of the work periods being of greater duration than the following rest period (McLean, 1992), similar values have been found in lower levels of the game (Deutsch, et al., 1996).

However, the current validity of this data is questionable because of the considerable changes the game has undergone over recent years, most notably the rule changes targeting the speed and continuity of the game. For example, in the 1991 World Cup final, the ball was in play for just 24 min. In contrast, Australian Rugby Union video analysts report that during the 2000 Super 12 (an elite Southern Hemisphere competition), the longest the ball was in play in a game was 44 min and 54 sec. The average was 37 min 30 sec, whereas the average during the 1999 Super 12 was 32 min (Cowden, 2000). In addition, the transition into the professional era has resulted in players devoting more time towards conditioning for the game and subsequently standards of play have been raised. Therefore, inferences regarding physiological load on players during rugby are most applicable from recent data collection.

Recent time-motion analysis of club and elite (Super 12) players (Deutsch, 2000), demonstrated that forwards perform over twice as much high intensity work as backs. Forwards spend 12% of total match time involved in 'high intensity' work, and backs ~ 5%. Overall, forwards were found to perform approximately 120 instances of work, with a mean duration of approximately 5 sec, separated by rest periods of ~ 35 sec. In addition, approximately 90% of the high intensity work performed by forwards comprises mauling, scrummaging, and tackling activities. These activities are highly specific to rugby forward play and require the rapid production of substantial

power. These researchers also measured HR and blood lactate in club players during matches. The HR data collected during this study revealed that a significantly greater proportion of time is spent above 85% HR_{max} by forwards (57% of the match) compared to backs (37%), presumably due to the involvement of forwards in frequent rucking, mauling, and scrummaging. Mean data for relative time greater than 95% HR_{max} was observed to range from 8% for outside backs, to 16% for loose forwards indicating a large anaerobic demand.

Measurements of blood lactate concentration ([BLa]) during match play currently represent the best available marker of the metabolic contribution of anaerobic glycolysis towards the physical demands of rugby. Blood lactate concentrations taken from players during a Scottish premier league match revealed a peak [BLa] range of 5.8–9.8 mmol.l⁻¹ from various individuals (McLean, 1992). These concentrations correlated with 56-85% of the peak blood lactate concentrations measured during a maximal treadmill test. Similar peak [BLa] values (8.8 mmol.l⁻¹) were recorded for front row forwards during colts (under 19 years) match play (Deutsch, et al., 1996). It is, however important to consider that because of the limitations of data collection during match play, samples were taken during convenient or predetermined times, therefore were not necessarily specific to passages of intense play. Furthermore, BLa is being metabolised (thus influencing net BLa accumulation) during periods of rest or low intensity exercise. These constraints suggest that current measurement techniques are likely to underestimate the true levels of peak [BLa]. Therefore, accurate assessment of the contribution of the anaerobic glycolytic system towards energy demands remains difficult.

Forward play is characterised by a greater volume and density of high intensity activity, with shorter recovery periods when compared to backs. The supply of energy to fuel the exercise pattern of forwards is

going to depend upon the dynamic utilisation of all the energy systems. Particularly the phosphagen system, as it contributes large amounts of energy towards intense exercise, especially during the initiation of movements. However, the replenishment of depleted phosphocreatine stores takes minutes rather than seconds (Bogdanis, et al., 1995). The short recovery periods available to forwards for recovery during a match (Deutsch, 2000) are unlikely to be of sufficient length to ensure complete resynthesis of phosphocreatine before the next bout of intensive activity is undertaken (Balsom, et al., 1992). Thus, anaerobic glycolysis represents a crucial energy system involved in the conversion of energy to meet the demands of forward play. Therefore it appears that forwards would be more likely than backs to benefit from a nutritional intervention designed to optimise pre-exercise muscle glycogen stores.

Our understanding of the physiological demands of rugby, and forward play in particular, is still very limited. Thus pinpointing specific mechanisms that cause fatigue, and subsequent strategies that may delay fatigue, will be an ongoing process. However, the available data reveal that rugby forward play is characterised by periods of very intensive and dynamic whole body activities interspersed with recovery periods of variable duration. This type of physical stress will result in dynamic utilisation of all the energy systems and, in particular, it will place large demands on the anaerobic glycolytic system.

Player characteristics

The physical, confrontational nature of rugby forward play places high demands for strength, power, and resilience from competitors. This generally predisposes a particular body-type (or shape) towards success in the sport. Therefore, rugby forward players tend to have a stereotypical set of anthropometric characteristics that enable them to

best meet the physiological demands of the sport. However it is noteworthy that some differences do exist between positional groups, most notably between hookers, props, and locks (Quarrie, et al., 1996).

Anthropometric studies of rugby forwards (Table 2.1) reveal that forwards tend to be taller in stature, of a larger mass, and with a higher percentage body fat than athletes involved in other MSS (Green and Bishop, 1976; Maughan, 1997). The unique physical characteristics of this group of athletes provides further grounds for research specific to rugby to be undertaken, rather than making inferences based on research concerning other athletic groups.

Table 2.1 Anthropometric data on rugby forwards

Position	n	Group	Height (cm)	Weight (kg)	%body fat
Forwards	9	Elite English ¹	184 (11.0)	100 (10.4)	-
Front row	5	International ²	190 (11.4)	103 (2.6)	16 (3.6)
Back row	6	English	185 (7.9)	101 (12.8)	12 (4.6)
Forwards	35	US elite ³	187 (7.2)	99 (9.1)	13 (3.9)
Forwards	8	1st grade club ⁴	181 (8.7)	88 (7.7)	12 (3.5)
Forwards	34	Elite English ⁵	182 (8.0)	90 (11.7)	13 (4.5)
Front row	5	First class ⁶	177 (3.7)	89 (3.7)	14 (2.1)
Second row	4	First class	197 (6.1)	101 (6.7)	11 (1.2)
Back row	5	First class	185 (5.9)	87 (6.8)	12 (2.5)
Forwards	15	Italian 1st class ⁷	184 (9.2)	96 (16.9)	-
Forwards	7	NZ 1st grade club ⁸	190 (4.6)	96 (4.3)	-
Forwards	12	NZ (elite) provincial	187 (5.5)	110 (6.2)	-
Forwards	15	SA 1st grade club ⁹	188 (8.3)	98 (8.7)	15 (2.7)
Mean (SD)					

- | | |
|-----|--------------------------------|
| No. | Reference |
| 1 | Holmyard and Hazeldine (1993) |
| 2 | Bell, Cobner, et al (1993) |
| 3 | Carlson, Carter, et al (1994) |
| 4 | Maud (1983) |
| 5 | Bell (1995) |
| 6 | Rigg and Reilly (1988) |
| 7 | Casagrande and Viviani (1993) |
| 8 | Deutsch (2000) |
| 9 | Jardine, Wiggins, et al (1988) |

Dietary Intakes

There has been little research investigating the usual nutrient intakes and dietary patterns of rugby players. However, limited data does exist from a survey of the 1991 Australian World Cup squad (Frail, 1993). This study estimated energy intake at 13.5 MJ (range 10 – 16.2 MJ), CHO intake at 4 g.kg⁻¹ (46% TE), and fat intake at 31% TE. All micronutrients were consumed in more than adequate amounts. As a group, dietary intake was lacking in CHO, high in fat and alcohol, and a protein intake on the high end of the recommended levels. Similar results have been reported in studies on Australian Rules football players (Burke, et al., 1991). A more recent report analysed the dietary practices of New Zealand academy forwards (Roberts, 1997). These players (n=5) had an energy intake of 12.4 ± 4.6 MJ and a CHO intake of 4.3 ± 1.8 g.kg⁻¹ per day. In general, the rugby population has been slow to adopt dietary habits that have been proven beneficial to endurance type sports (Frail, 1993; Roberts, 1997).

Fatigue in rugby forward play

Fatigue can be broadly defined as the failure to maintain the required or expected force or power output (Gibson and Edwards, 1985). Yet when we consider the numerous movement tasks presented to a forward within the team pattern of a rugby match, it becomes immediately obvious that players are going to be exposed to various sources of fatigue throughout a match. One example would be when players are involved in repeated scrums, in this situation players might be required to perform a static or dynamic pushing movement for 2-8 sec, then repeat successively, often without adequate time to recover. Another example is when players are required to quickly cover a considerable amount of distance before performing a high power; skill

orientated movement such as mauling or tackling. Thus, it is important to recognise that a dietary manipulation targeted at maximising muscle glycogen stores may influence only one of these fatigue mechanisms. For example, at higher intensities ($>90\% \dot{V}O_2 \text{ max.}$) of exercise a greater proportion of the energy requirement is covered by anaerobic processes. This results in accumulation of metabolic end products and ultimately a perturbation of cellular homeostasis. At these high intensities, fatigue is characterised by a marked depletion of high-energy phosphates and a decrease in pH, due to lactate accumulation, yet adequate muscle glycogen levels. Therefore, fatigue in this instance might be relatively transient compared to fatigue caused by depletion of energy substrate stores. There has also been speculation that a diet low in CHO may induce metabolic acidosis which may reduce the capacity to sustain high intensity exercise over a short (~ 5 min) duration (Maughan, et al., 1997). In addition, several steps in the contractile process are known to be energy dependant such as: maintenance of the $\text{Na}^+\text{-K}^+$ gradient over the sarcolemma; re-uptake of Ca^{2+} by the sarcoplasmic reticulum (SR); cross bridge cycling; and coupling between t-tubule depolarisation and Ca^{2+} release from the SR. Investigation, during match play, into the role these fatigue mechanisms may play in rugby performance is difficult, if not impossible, with currently available techniques.

Performance in a brief sprint does not appear to be significantly altered by endogenous carbohydrate status (Cheetham and Williams, 1987). However, the work of Boobis et al. (1982) has demonstrated that up to 25% of pre-exercise muscle glycogen can be depleted following a 30 sec cycle sprint. Therefore, if single sprints (in addition to other movement modalities) were undertaken successively in an intermittent fashion it is feasible that muscle glycogen levels may eventually limit an individual's ability to sustain the high power output required for optimal performance. This contention is not supported by the levels of total muscle glycogen recorded in club players following

match play (Jardine, et al., 1988). However, total muscle glycogen measurements do not discriminate between selective use of glycogen in different fibre types, nor was performance measured during Jardine's study.

Friden, Seger, and Ekblom (1989) showed differential depletion of glycogen from different fiber types as well as specific intramuscular compartments (e.g., subsarcolemmal, intermyofibrillar) after 60 bouts of intermittent supramaximal exercise (8 sec at 200% of $\dot{V}O_{2\max}$ with 52 sec rest between each bout). These depots may be called upon differently, depending on the intensity and duration of the exercise. Therefore, a more detailed study of the fibre type pattern of muscle glycogen utilisation in rugby would be of interest to determine if localised depletion of specific glycogen depots contributes towards fatigue.

Pizza, Flynn, et al. (1995) used a randomised, crossover design to examine the effect of a CHO loading regimen on high intensity, short duration run performance. Eight trained runners completed a 15 min sub-maximal run (75% of $\dot{V}O_{2\max}$) and a performance run (at $\dot{V}O_{2\max}$ i.e. 15.5 km.h⁻¹, 3.3 ± 0.6% grade) after two dietary treatments – mixed diet (LCHO) which contained ~4 g.kg⁻¹ and high CHO diet (HCHO) which contained ~8 g.kg⁻¹. Six of the eight subjects increased their time to exhaustion (p=0.03) after the HCHO treatment. The authors concluded that because of the increased involvement of type IIb muscle fibers during high intensity exercise, and in light of the available evidence of glycogen utilisation in type IIb muscle fibers, it is possible that the increased time to exhaustion for HCHO was the result of supercompensated glycogen levels in type IIb muscle fibers (Greenhaff, et al., 1991; Maughan and Poole, 1981). But an individual's capacity to perform maximal exercise of short duration can be limited by many factors, including intramuscular acidosis, depletion of

phosphocreatine stores, and glycogen depletion of type IIb muscle fibers (Greenhaff, et al., 1991). This study highlights the complexity of fatigue during high intensity exercise.

Summary

There are a limited number of well-controlled studies investigating rugby and the potential relationship between pre-exercise CHO intake, muscle glycogen, and performance. The modern game of rugby union, and specifically forward play, places a wide range of physiological demands on players. Players are required to repeatedly perform skilled movements that require large amounts of power. Furthermore, the confrontational nature of rugby dictates that these movement patterns are very dynamic and often occur in pressure (both physical and cognitive) situations. Therefore, a large muscle mass is essential for success, but this must be balanced against the requirement for mobility since the game is played at pace, over a large playing area.

The aim of the present study was to examine the effects of three days of a carbohydrate rich diet ($>8 \text{ g.kg}^{-1}$) vs three days of a lower carbohydrate diet ($<4 \text{ g.kg}^{-1}$), on muscle glycogen levels, and on subsequent performance in an 80 min rugby simulation.

3 Methods

Study design

A randomised, blind, crossover design was used in which subjects consumed either a low carbohydrate (LCHO) or high carbohydrate (HCHO) diet for three days prior to testing (Figure 3.1). Upon entry into the study all subjects gave full informed consent, completed a medical history questionnaire, and recorded their usual diet using a four day weighed diet record (Appendices A, B, and C). Subjects also performed a $\dot{V}O_2$ max test. A small payment was offered to each subject to partially compensate for time committed to the study. Seven days separated each testing day; the entire data collection phase took less than three months. The principal researcher took several anthropometric measures (height, weight, S6SF). All subjects were familiarised with the rugby simulation circuit (simulation) before testing. This study received ethical approval from the Southern Regional Health Authority ethics committee (Dunedin, New Zealand).

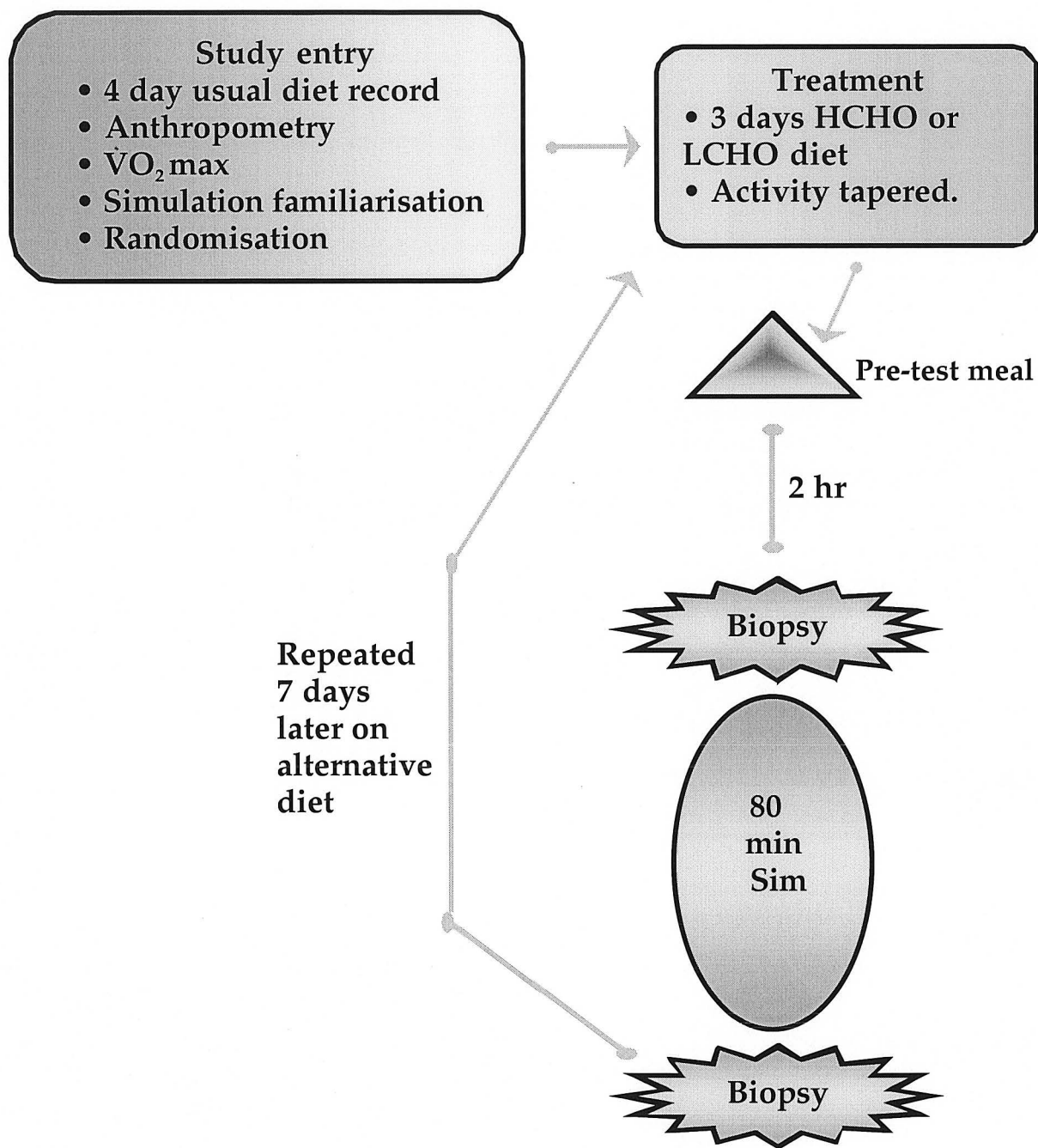


Figure 3.1 Overview of study design

Subject characteristics

Twelve male, premier, club rugby players volunteered to participate in this study during the pre-season and early competition season. They were required to be injury free, involved in preseason rugby fitness conditioning, and meet the positional criteria of being a loose forward, lock, or hooker. Two subjects were unable to complete the entire study. One sustained an injury during a match between testing days, while the other suffered from influenza during the final testing session. Their data were not included in the analyses.

Anthropometry

Body composition was assessed upon entry into the study by the principal researcher. Height (stadiometer; Holtain Ltd., UK), weight (balance; Seca alpha 770, Germany), and sum of six skinfolds (0.2 mm Holtain Ltd., UK) (triceps, subscapular, supraspinatus, abdomen, thigh, medial calf) were measured according to the techniques used for the LINZ (1993) survey (Wilson, et al., 1993).

Maximal aerobic capacity ($\dot{V}O_{2\max}$) test

An incremental $\dot{V}O_{2\max}$ protocol was performed on a treadmill (Model 24-72, Quinton Instrument Company, USA) with oxygen consumption measured from expired air via a metabolic cart (Sensormedics Ltd. USA). Subjects warmed up at 10 km.hr⁻¹ for 5 min before beginning the test at a speed of 12 km.hr⁻¹; this speed was maintained for 2 min and then speed was increased by 1 km.hr⁻¹ every 2 min until a speed of 15 km.hr⁻¹ was achieved. After completing 2 min at

15 km.hr⁻¹, the elevation of the treadmill was increased by 1 degree every minute until subjects failed to maintain the prescribed pace.

Dietary interventions

The diets for this study were required to supply CHO at two levels of intake, either <4 g.kg⁻¹ (LCHO), or >8 g.kg⁻¹ (HCHO). To facilitate the construction of diets with a “usual” eating pattern, CHO dense foods were identified to facilitate consumption of CHO in large quantities. Similarly, foods high in fat and protein were identified for use in the LCHO diet. The absolute amount of CHO, in grams, at the two dietary levels, was calculated for each specific body weight.

Both treatment diets were designed for three meals and three snacks throughout the day, thus ensuring an even spread of CHO intake. Sample menus were provided and converted to individually quantified diet and instruction sheets (Appendix D) with the required amounts (adjusted to the body weight of the subject) of each food entered on the sheet. On the HCHO diet the most important instruction was to consume at least the stipulated amount of CHO, whereas on the LCHO diet the instruction emphasised was not to consume more than the stipulated amount of CHO.

Pre-simulation meal

A standardised meal was consumed two hours before each simulation. The meal consisted of 44 g of Complian™, a banana (120-130 g), and 1000 ml of water. The nutrient composition of this meal was 1600 kJ, 65% of total energy (TE) from CHO, 15% TE from protein, and 20% TE from fat.

Simulation

Simulation development

The simulation was designed to mimic (within an ethical research setting) the physical demands of forward play during club (premier) match play. The simulation was based on time-motion analysis data collected during the 1996 and 1997 seasons. Thirty-seven forwards were filmed at least twice during club matches and Super 12 matches. Detailed time motion analysis was performed on match tapes to determine movement patterns and exercise density profiles for each position (Deutsch, 2000). These data were pooled by similar position and subsequently used to design a simulation circuit (Figure 3.2).

Simulation design

The simulation instrument provided the controlled environment necessary to objectively test the efficacy of a specific dietary intervention (i.e. pre-exercise CHO manipulation) on rugby performance parameters and muscle glycogen metabolism. Some of the more physical components of rugby were not included because of ethical constraints. A trained assistant guided each subject through the simulation. The simulation was located in an indoor tennis arena on a sand based 'Astro turf' surface. Each lap of the simulation took approximately 11 min to complete and involved most elements of forward play including scrummaging, mauling, sprinting, jogging, cruising, walking, tackling, and standing still. As per match play, a five min break was enforced after 40 min of the simulation to mimic half-time during games. This occurred during lap 4, subsequently first-half performance was based on laps 1-3 and second-half performance on laps 5-7. Players were permitted to consume water ad libitum at the end of each lap. A mean total of ~750 mls was consumed by each subject during each simulation trial.

Familiarisation

Upon entry to the study each subject performed a 'walk through' the simulation to gain an understanding of the test instrument. One week prior to data collection subjects completed a lap at 80% of maximal intensity followed by four laps at 100% of maximal intensity. Performance based feedback was given to the subjects' to ensure they learned the skilled components of the simulation.

Performance and physiological load measurement

Continuous HR (5 s intervals) was recorded via a short-range telemetry system (Sports tester PE₃₀₀₀, Polar Electro Oy, Kempee, Finland) during the entire simulation. The HR data collected was grouped into relative time spent within specific HR zones (<75% HR_{max}, 75-85% HR_{max}, 85-95% HR_{max}, >95% HR_{max}). Maximal HR was taken as the highest recorded during match play, simulation testing, or during the VO₂max test.

Blood samples were taken from the earlobe of each subject prior to the simulation (baseline) at the completion of every second lap, at half time, and at full time. Samples were analysed (Accusport, Boehringer, Mannheim, Germany) immediately to determine blood lactate concentration ([BLa]).

Several performance measurements were taken during each lap of the simulation. These included:

- 1) Score 25 m Sprint = Timed 25 m sprint using opto-reflective timing lights (School of Physical Education, University of Otago, NZ).
- 2) Score ruck = Mean force output during five simulated rucking/mauling pushing movements lasting 2, 4, 4, 5, and 6 sec.

- 3) Score scrum = Mean isometric force during four simulated scrummaging movements (2-8 sec).

Mean force output for simulated mauling was measured using the Grunt 2000 mauling ergometer (E-type Engineering, Invercargill, NZ). Briefly, subjects pushed a trolley weighing approximately 130 kg against the resistance of an attached bungee cord, for a designated time. The tension force of pushing was measured using a load cell (electronics workshop, School of Physical Education, Dunedin, NZ). The load cell was connected to a personal computer for data calculation and storage.

Mean force output during simulated scrummaging was calculated as mean peak force (N) produced during maximal isometric contractions (~2 sec). Force (N) (tension) was measured along a rigid line attached to a load cell, amplifier, and personal computer as above.

Performance measures during these tests were combined to form a 'Performance score' for each lap of the simulation. Performance on each task was judged in relation to individual maximal values obtained during the experiment. This provides a total weighted score (TWS) for each lap of the simulation. Each performance was calculated according to the following formula:

$$\text{Performance score} = 1 - \frac{(\text{Personal best} - \text{Performance measure})}{\text{Personal best}}$$

The relative weighting of these activities (based on their relative contribution to match play) was calculated from match play data (Deutsch, 2000). The following equation was used to calculate the TWS for each lap of the simulation:

$$\begin{aligned} \text{TWS} = & 5.19 (\text{Score 25 m sprint}) + 3.35 (\text{Score maul 2 sec}) + 6.7 (\text{Score} \\ & \text{maul 4 sec}) + 6.71 (\text{Score maul 4 sec}) + 6.71 (\text{Score maul 5 sec}) \\ & + 10.07 (\text{Score maul 6 sec}) + 5.45 (\text{Score scrum 2 sec}) + 10.90 \\ & (\text{Score scrum 4 sec}) + 16.36 (\text{Score scrum 6 sec}) + 21.81 (\text{Score} \\ & \text{scrum 8 sec}) \end{aligned}$$

The performance results were also split into first-half (laps 1-3), and second-half (laps 5-7) scores to determine if the subjects fatigued during the second half following either dietary treatment.

Dietary analysis

Upon entering the study subjects completed a four-day weighed (Salter – Sensor 2050 electronic scales, Kent, England) diet record (Appendix C). During the HCHO and LCHO treatments three-day weighed diet records were also completed. Each diet record was analysed (Diet Cruncher 2; Nutricomp, NZ) for nutrient composition by the principal researcher.

Muscle biopsies

An experienced sports physician took all muscle tissue samples from the vastus lateralis using the percutaneous needle biopsy technique previously described (Bergstrom, 1962; Evans, et al., 1982). Muscle biopsies were taken before each trial. A pre-simulation biopsy (2 hrs after the standardised breakfast), and a post-simulation biopsy were taken from the same incision. Care was taken with the angle of the biopsy needle during post-simulation sampling to ensure that tissue near the pre-simulation sample site was avoided. Alternate legs were used for each trial. Excess connective tissue and blood were removed from the samples. One half of the sample was mounted (Cryoform,

Catalogue No 3383, International Equipment Company, Needham. MA 02194, USA) longitudinally on cork for muscle fibre type specific analysis. The remaining tissue was then split into equal pieces (for duplicate analysis) and placed into cryovials. All samples were then frozen in -90°C liquid nitrogen and stored in a -80°C freezer for later analysis. All participants took part in team training sessions the day after testing, and all biopsy incisions healed without infection or complications.

Muscle glycogen analysis

The unmounted muscle samples were weighed (Model AA-200; Denver Instrument Company, USA) and then homogenised by adding 1 ml of 1M NaOH and placed in a water-bath and vortexed periodically for 10 min or until digestion was complete. The samples were allowed to cool before adding 267 μl of 1.5M citric acid and vortexed again. From this homogenate two 500 μl samples were taken and 50 μl of amyloglucosidase enzyme (Boehringer Mannheim cat no. 102 857) was added to each. Samples were vortexed again before being incubated in a 45°C water-bath overnight. Each sample was then vortexed and filtered using 0.45 μl filter paper.

The resulting glucose was analysed on the Cobas Fara 2 using a Boehringer Mannheim test kit for D-glucose (art. 716 251). The intra-assay coefficient of variation was 0.8% for the glycogen analysis and the calculated recovery was $99.1 \pm 3.6\%$, using a glycogen standard (Sigma Rabbit liver glycogen type III).

Unfortunately the muscle fibre type specific analysis was unable to be carried out as all samples rapidly thawed following the malfunction

of the -80°C freezer. This represents a significant loss to the original goal of this study, but does represent opportunities for future research.

Statistical analysis

The data were analysed using paired t-test: or as a cross over trial using ANOVA as appropriate. In order to investigate the effect of diet on the TWS score, the mean of the first three lap scores was compared with that of the last three. Differences with 95% confidence intervals (CI) are presented for the treatment effects. The model included an interaction effect between treatment, and game halves.

4 Results

Subject characteristics

Subjects who met the selection criteria of being injury free, currently playing premier grade rugby and playing in the 'forwards' were young male adults, of large mass, and of tall stature (Table 4.1). Skinfold measurements (S6SF) revealed moderate subcutaneous fat values with a large variation between individuals. Measurements of aerobic capacity revealed a moderate relative capacity and impressive absolute aerobic capacity

Table 4.1 Subject Characteristics, (n=10)

	Mean (SD)	Min	Max
Age (yrs)	23 (4)	20	31
Mass (kg)	96 (9)	91	110
Height (cm)	189 (7)	175	197
S6SF (mm)	75 (40)	35	147
VO ₂ (L.min ⁻¹)	5218 (396)	4394	5729
VO ₂ max (ml.min ⁻¹ .kg ⁻¹)	55 (6)	45.8	61.5

Dietary treatments

Subjects displayed good adherence to the prescribed dietary regimens. The treatment goals of consuming >8 g.kg⁻¹ bodyweight (HCHO) and <4 g.kg⁻¹ bodyweight (LCHO) were achieved (Table 4.2).

Significantly ($p=0.02$) greater energy was consumed on the HCHO treatment compared to the LCHO treatment (Table 4.2). However, the energy intake on the LCHO treatment was equivalent to that observed in subjects on their usual diets, thus, there was no energy deficiency on the LCHO treatment but rather an energy excess on the HCHO treatment.

Protein intake was adequate ($1.4\text{--}2.0\text{ g.kg}^{-1}$) for all treatments but did vary, with the LCHO treatment providing significantly more ($p=0.005$) than the HCHO treatment. The largest difference in macronutrient intake between the two dietary treatments was observed for fat intake. The LCHO treatment provided significantly ($p=0.005$) more dietary fat, this fat intake also provided more of the total energy consumed during that treatment (Table 4.2).

Table 4.2 Dietary treatment composition, (n=10)

Nutrient	Diet treatment		
	Usual	HCHO	LCHO
Energy (MJ)	14.6 (2.1)	18.6 (4.2)	14.9 (3.3)*
CHO (g)	441 (113)	784 (242)	241 (56) [†]
(% TE)	47.7 (6.4)	66.4 (6.4)	26.7 (3.4) [†]
(g.kg ⁻¹)	4.5 (1.2)	8.3 (2.6)	2.5 (0.6) [†]
Protein (g)	133 (12)	147 (21)	193 (43) [†]
(% TE)	15.6 (2.2)	13.7 (2.1)	22.3 (3.3) [†]
(g.kg ⁻¹)	1.4 (0.2)	1.5 (0.2)	2.0 (0.5) [†]
Total Fat (g)	120 (24)	75 (14)	200 (52) [†]
(% TE)	30.4 (4.9)	15.2 (3.4)	49.6 (3.6) [†]
Dietary Fibre (g)	30 (8)	44 (12)	26 (7) [†]
Mean (SD), * $p=0.02$, [†] $p=0.005$			

Simulation characteristics

Activity patterns

The simulation comprised several different movement modalities, specifically movements which are important components of rugby forward play (Figure 4.1). The majority of the time was spent walking or inactive (standing still). Other movements such as mauling, scrummaging, and cruising were also prominent. High intensity movements, such as sprinting and tackling were less frequent.

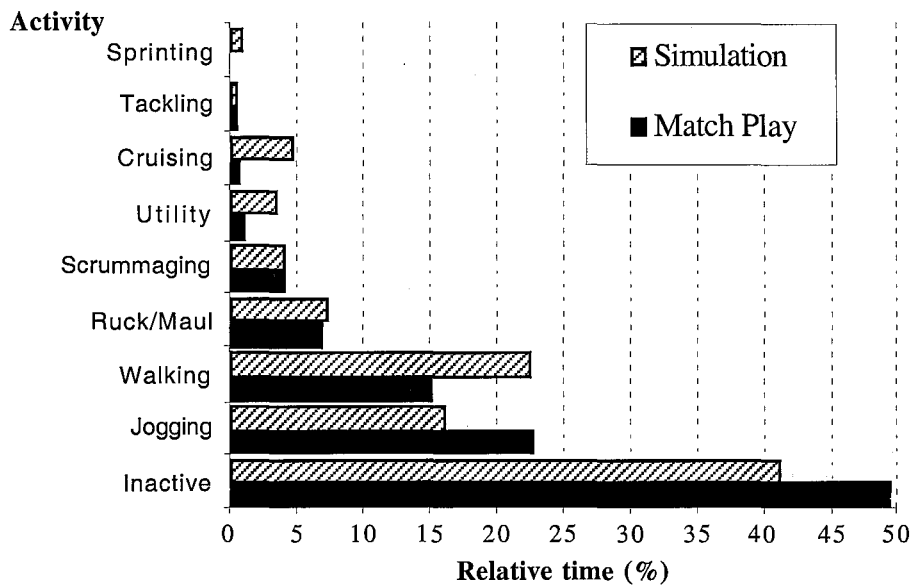


Figure 4.1 Relative time spent performing defined activities during match play (club-premier) (Deutsch, 2000) and the simulation

Work: Rest ratios

The simulation resulted in a large amount of the total time (~30%) with a W:R ratio of 1:12 (Figure 4.2). The simulation did not provide exercise stimulus within the W:R ratio of 1:11 to 1:5. However the W:R ratios of 1:4 through to 3:1 were prominent.

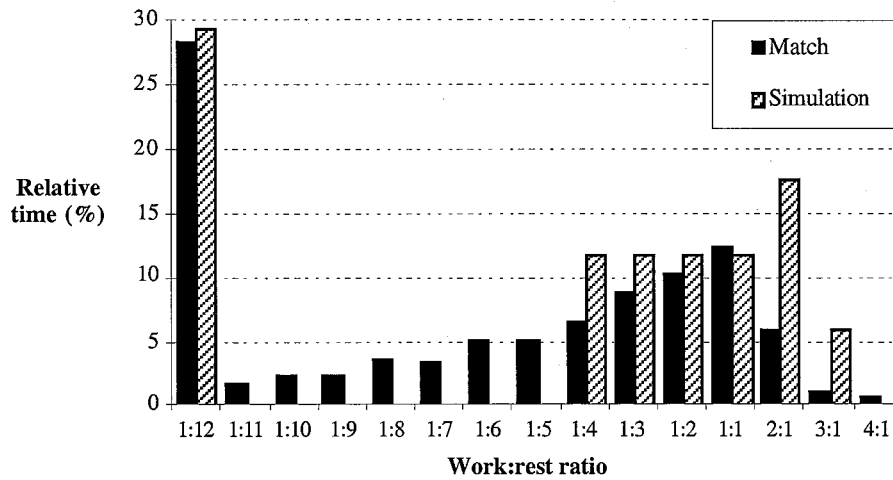


Figure 4.2 Relative time spent exercising at each W:R ratio during the simulation and match (club-premier) play (Deutsch, 2000)

Heart rate

Continuous HR monitoring during the simulation revealed similar profiles between the two treatments (Figure 4.3). During the simulation, subjects spent a relatively small amount of time (<5%) exercising with HRs greater than 95% of their HR_{max} . In contrast, the subjects spent most of the simulation (~40%) with HRs of 85-95% of HR_{max} . The remaining time was spent with HR in the lower zones of 75-85% and <75% of HR_{max} .

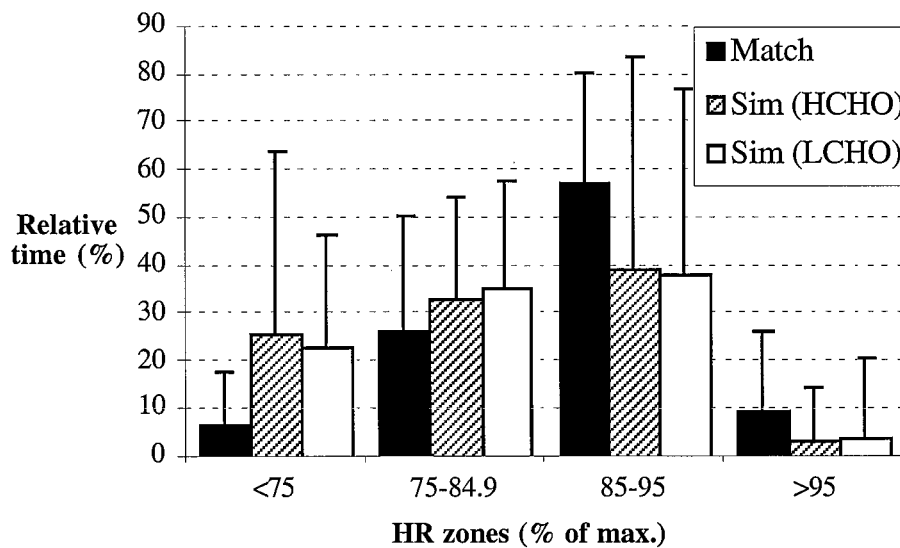


Figure 4.3 Relative time spent by forwards within each HR zone for the 80 min simulation (HCHO and LCHO treatments) compared to match (club-premier) play (Deutsch, 2000)

Blood lactate

Blood lactate samples were collected before the simulation (baseline), at the completion of Laps 2, 5, and at half time and full-time (Figure 4.4). Blood lactate concentrations were not significantly different between treatments. A peak blood lactate concentration of $4.1 \pm 1.6 \text{ mmol.l}^{-1}$ was reached for the HCHO treatment. A similar value of $4.0 \pm 1.1 \text{ mmol.l}^{-1}$ was reached for the LCHO treatment. Both of these results occurred at the finish of the simulation.

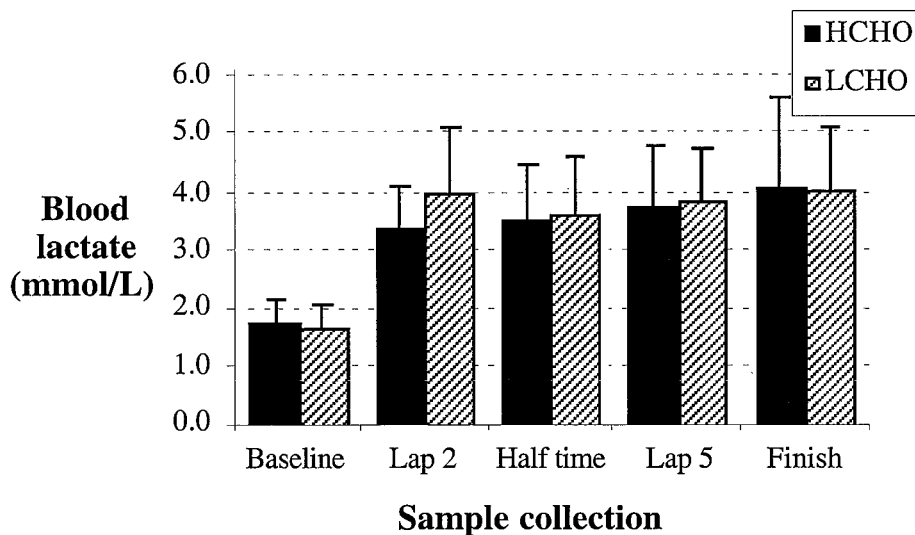


Figure 4.4 Mean (SD) blood lactate concentrations (mmol.l^{-1}) during the simulation following HCHO and LCHO dietary treatments, $n=10$

Muscle glycogen

Subjects exhibited a significantly higher ($p=0.002$) pre-simulation muscle glycogen concentration following the HCHO treatment compared to the LCHO treatment (Table 4.3). Muscle glycogen utilisation was approximately 50% of the available stores for both treatments, therefore, the amount used during the HCHO treatment was significantly higher ($p=0.01$) than during the LCHO treatment. Neither condition resulted in total depletion of muscle glycogen stores post-simulation, although during the LCHO treatment the subjects did get to a low level. The results are also presented as a box and whisker plot below (figure 4.5).

Table 4.3 Mean (SD) muscle glycogen concentration in biopsy samples following HCHO and LCHO treatments with samples collected prior to (pre) and after (post) the simulation

Treatment	Muscle glycogen concentration (Glucosyl units, mmol.kg ⁻¹ ww)			
	HCHO	LCHO	difference	95% CI
Pre-Sim.	102.4 (28.2)	59.1 (17.6)	43.8 [†]	(19.4, 68.2)
Post-Sim.	50.9 (26.5)	26.0 (9.5)	25.0 [*]	(4.2, 45.8)
difference	51.4 (13.3)	33.1 (14.7)	18.8 ^{**}	(4.3, 33.3)

† $p=0.002$, * $p=0.02$, ** $p=0.01$, CI (confidence intervals) adjusted for order.

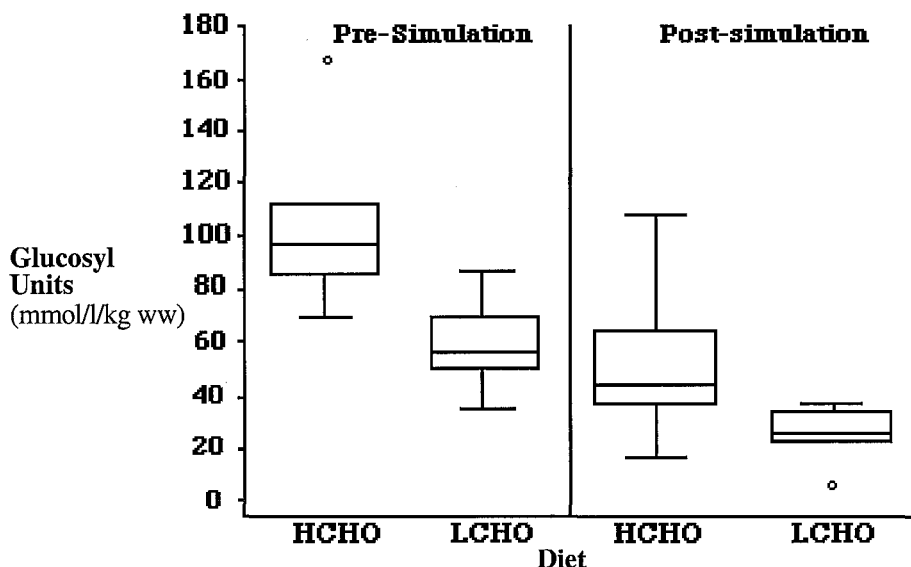


Figure 4.5 Box and whisker plot of muscle glycogen concentration (Glucosyl units) for HCHO and LCHO dietary condition pre- and post-simulation

Simulation performance

Figure 4.6 illustrates differences in performance (TWS). A positive TWS (%) difference signifies superior performance following the HCHO treatment compared to the LCHO treatment.

The results demonstrate a small negative difference between the treatments after lap 1. The TWS difference was still slightly negative for lap 2 without reaching significance. Laps 3–7 all exhibited a positive TWS difference between the treatments. This strong positive trend was most pronounced during laps 4 and 5. Individual TWS results are presented in Appendix E.

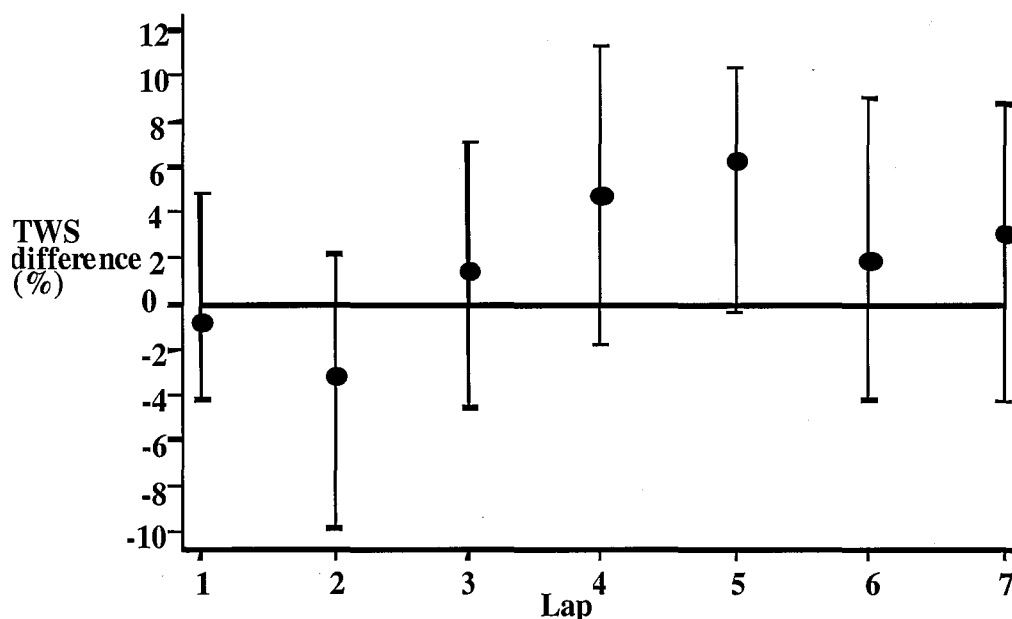


Figure 4.6 Difference (%) between treatments (HCHO - LCHO) in simulation performance (TWS) with associated 95% confidence intervals

A large decrease in performance from the first-half (laps 1-3) to the second-half (laps 5-7) of the simulation was observed following the LCHO treatment (Table 4.4). This trend was not found following the HCHO treatment. The interaction effect between treatment, and the performance decrease from the first-half to the second-half, narrowly failed to reach significance ($p=0.08$).

Table 4.4 Mean (SD) TWS for first-half (laps 1-3) and second half (laps 5-7) for the HCHO, and LCHO treatment

Treatment	TWS		
	First half	Second half	Diff. (95% CI)
HCHO	89.2 (4.6)	88.6 (4.9)	0.6 (-3.1, 4.3)
LCHO	89.6 (6.8)	85.0 (6.4)	4.6 (1.7, 7.6)

* interaction effect between treatment and game halves, $p=0.08$; 0.7 effect size

5 Discussion

Key findings

The purpose of this study was to determine the effects of three days of a high CHO diet ($>8 \text{ g}\cdot\text{kg}^{-1}$) versus three days of a lower CHO diet ($<4 \text{ g}\cdot\text{kg}^{-1}$), firstly, on muscle glycogen storage and metabolism, and subsequently, on performance in an 80 min rugby simulation.

The participants achieved the dietary treatment objectives and consumed a significantly greater amount of CHO during the HCHO treatment compared to the LCHO treatment. Undergoing this dietary manipulation caused a significantly greater pre-exercise muscle glycogen concentration following the HCHO treatment compared to the LCHO treatment. Therefore, an acute three-day dietary intervention is capable of significantly altering the pre-exercise muscle glycogen concentration in rugby forwards.

Subjects' performance (as measured by the TWS) during the simulation with an elevated muscle glycogen store (i.e. following the HCHO treatment) was superior compared to following the LCHO treatment. The difference in the TWS from the first-half to the second-half between the two treatments was 4.0 units (or 4.5%) with the interaction effect between treatment and game halves narrowly failing to attain statistical significance ($p=0.08$). This observed trend for a decrease in simulation performance following the LCHO treatment and

the maintenance of performance after the HCHO treatment may however, may be of practical significance, especially considering the small sample size of this study.

The simulation produced a lower physiological response than that measured during matches, however, it did place sufficient physiological demand on the subjects to cause a decrease in muscle glycogen stores of ~50% of their initial value for both treatments. Therefore, the higher initial muscle glycogen concentration attained after the HCHO treatment resulted in a significantly greater absolute use of stored glycogen (i.e., increased utilisation). Post-exercise muscle glycogen concentrations did not totally deplete in either dietary treatment, although a low muscle glycogen concentration was observed following the LCHO treatment. In order to correctly interpret the results it is important to firstly discuss the merits of the simulation as a testing instrument for forwards in rugby.

Simulation characteristics

To determine the actual efficacy of a dietary manipulation on performance it is necessary to go beyond the confines of the laboratory and perform field-based research. In doing so, the external validity of the study will be enhanced with the concomitant risk of losing elements of the internal validity. Such was the task for this study, designed with the intention of providing an appropriate mix of these two crucial research components.

This study represents the first known attempt at simulating the physical demands of rugby forward play, and the first study to use a simulation as an instrument to test the efficacy of a nutritional intervention in rugby. Accurate simulation of the physiological demands

of forward play presents an extremely challenging research task. Therefore determining the precision and reliability of the simulation instrument are key ingredients of this study.

Comparisons between recent match play (Deutsch, 2000) and the simulation reveals that the simulation provides similar, or more demanding W:R ratios to that of a match. During the simulation, subjects spend a similar relative amount of time (29%) with a W:R ratio of 1:12 compared to match play results (28%). The simulation is not designed to provide the W:R ratios of 1:11–1:5 and therefore no part of the simulation matches the small amount of time (1-5%) these W:R ratios contributes to the total exercise period of match play. Subjects spend approximately 12% of their time with in the W:R ratio of 1:4-1:1 during the simulation, similar values are observed during match play. A large difference is noted for the time spent exercising at 2:1 W:R ratio with the simulation producing 18% compared to 6% during recorded matches. In addition, the simulation demands more time with a ratio of 3:1 than during a match (6% compared to 1%).

Activity pattern levels during the simulation also closely resemble those recorded during match play (Deutsch, 2000). Subjects spend 0.8% of the simulation sprinting compared to 0.1% during match play. Similar periods of time for the simulation compared to the match is spent performing the activities: tackling, scrummaging, mauling, and utility movements. During match play, players spend only 0.6% of their time cruising compared to 5% during the simulation. Subjects spend 22% of the simulation walking compared to 15% of match play. Jogging is also a predominant match play activity comprising 23% of total time compared to 16% during the simulation. Finally, subjects are inactive for 41% of the time during the simulation compared to 49% of time during match play.

Despite activity and work density patterns (W:R ratios) being similar between the simulation and matches, one of the physiological stress markers, HR response, was lower during the simulation than that observed during match play. Match play resulted in a greater amount of time in the HR zones 85-95% of HR_{max} and >95% of HR_{max} compared to the simulation (58% and 10% compared to 39% and 3%, respectively). Consequently, less time was spent within the HR zones <75% of HR_{max} and 75-85% of HR_{max} . This difference was particularly pronounced for <75% of HR_{max} where the simulation resulted in subjects spending ~24% of their relative time within this zone compared to ~6% during match play (Deutsch, 2000). This is an interesting finding considering the movement modalities were similar according to time motion analyses, and total work time was almost identical in the simulation compared to the match play analyses. It is speculated the psycho-physical stress of competition and an increase in catecholamine release may account partially towards this difference. The principle causal factor appears to be a failure of the high intensity components of the simulation instrument to match the demands of the competitive environment.

Another marker of physiological stress used in this study was [BLa]. Blood lactate concentrations recorded during the simulation did not reach the levels measured during match play (Deutsch, et al., 1996; McLean, 1992). Several factors are most likely contributing towards this. The simulation did not provide as much vigorous upper body work as experienced during match play. Thus, greater blood volume is available for fueling the lower extremities during the simulation compared to match play. This would result in some of high intensity components of the simulation not truly representing the whole body demands of forward play specific movements (eg, mauling, scrummaging). In addition, sampling for lactate was done at specific locations on the circuit (i.e. end of Lap 2 and 5) or specific times (half-

time and full-time). These sampling times may not have coincided with the peak blood lactate levels experienced by the subjects.

Therefore, it appears that the simulation may underestimate the true physical demands of forward play, even at club level. There are several reasons why this may be occurring. Firstly, certain elements of the game are extremely difficult to emulate within the confines of a research setting. For example, the absence of confrontational physical contact during the simulation (an ethical decision) decreases the danger and intimidation aspect of the sport. Therefore, the level of sympathetic nervous system activation experienced during a non-competitive simulation is unlikely to approach levels experienced during a game. Furthermore, it was not possible, with the current simulation; to emulate the multi-directional forces encountered by players during a game. In addition, many of the subtle movements not accounted for in time-motion analysis of match play (e.g., unexpected changes in direction, constantly being put off balance by opponents) were difficult to include in the simulation instrument. Therefore, the simulation contained a greater number of movement patterns with a more predictable nature, compared to that experienced in match play.

In summary, while the simulation provided similar physical demands to those experienced during a match, based on the physiological markers of exercise stress used in this study (HR and [BLa]), the simulation was not as intense as a match. This has implications for the interpretation of the muscle glycogen, and performance results. It can be speculated that muscle glycogen utilisation will be greater during a match compared to the simulation, and the observed performance decrease in the latter half of the simulation may underestimate match play fatigue.

Dietary treatments

The design of the dietary treatment regimens aimed, to as close as possible, resemble the usual eating patterns of young male rugby players. Therefore, care was taken to include everyday foods into the dietary prescription. Subjects demonstrated good adherence to the prescribed dietary treatments and the CHO targets were achieved for both the LCHO treatment ($<4 \text{ g.kg}^{-1}$), and the HCHO treatment ($>8 \text{ g.kg}^{-1}$). Unfortunately, the total energy intakes were not matched during the LCHO and HCHO treatments. While consuming the HCHO treatment, subjects were encouraged to eat freely from of CHO rich food list to facilitate the consumption of the targeted amount of CHO per day. This may have contributed to subjects consuming a significantly greater energy intake ($18.6 \pm 4.2 \text{ MJ}$) during the HCHO treatment compared to the LCHO treatment ($14.9 \pm 3.3 \text{ MJ}$). However, it is important to note that the usual dietary intake (measured on entry to the study) was similar ($14.6 \pm 2.1 \text{ MJ}$) to the intake during the LCHO treatment. Therefore, it is unlikely that subjects were consuming insufficient energy during either treatment period. Furthermore, there is no evidence that consuming additional energy above the usual intake will acutely affect performance in high intensity, intermittent exercise. However, because the maintenance of an ideal bodyweight can be an ongoing concern for some players the habitual intake of a CHO rich diet, without adequate control of energy intake, may have implications for long term weight control (Frail, 1993). Finally, these energy intake levels are similar to those recorded by other team sport athletes and therefore it is assumed that the subjects did not under-report or under-eat during the recording of their usual diet or during each dietary treatment (Burke, et al., 1991; Roberts, 1997; van Erp-Baart, et al., 1989).

Muscle glycogen

After the three-day HCHO dietary treatment and standardised breakfast, subjects had a mean muscle glycogen concentration of $102.4 \pm 28.2 \text{ mmol.kg}^{-1} \text{ ww}$. This was significantly higher than the mean LCHO pre-simulation muscle glycogen concentration of $59.2 \pm 17.6 \text{ mmol.kg}^{-1} \text{ ww}$. These glycogen levels are not appreciably higher than those recorded in untrained people on a mixed diet and approximately 40% lower than measured in endurance trained runners under similar dietary conditions (Bergstrom, et al., 1972; Costill and Miller, 1980; Sherman, et al., 1983; Sherman, et al., 1981). Similar pre-exercise muscle glycogen levels have been observed for club players under normal and CHO-loaded conditions (Jardine, et al., 1988).

The pre-simulation muscle glycogen levels might have been influenced by the degree of rest (or tapering) during the dietary treatment period. This study coincided with pre-season training and games, therefore team commitments may have, at times, prevented complete rest during the three dietary treatments days. These factors may have collectively contributed towards the observed variance in muscle glycogen concentration.

The post-simulation glycogen levels reveal that subjects utilise significantly more muscle glycogen when they have a higher initial glycogen level due to increased CHO intake. The observed relationship between these two variables has been found in several other investigations (Bangsbo, et al., 1992a; Jardine, et al., 1988). However, this is not a consistent finding and a relationship between initial muscle glycogen levels, and subsequent utilisation may not necessarily translate to an improvement in performance, or delay in fatigue in MSS.

The post-simulation muscle glycogen levels were not totally depleted with either treatment, yet following the LCHO treatment muscle glycogen concentration did fall to $26.0 \pm 9.5 \text{ mmol.kg}^{-1} \text{ ww}$. This approaches the proposed threshold of $\sim 25 \text{ mmol.kg}^{-1} \text{ ww}$ at which the onset of fatigue has been observed during prolonged ($>90 \text{ min}$) moderate-intensity exercise at a fixed exercise intensity of 70-75% $\dot{V}\text{O}_2 \text{ max}$ (Bergstrom, et al., 1967; Bosch, et al., 1994). It is possible however, that this level of glycogen may have compromised the ability of subjects to perform the powerful movements required to maintain the TWS late in the simulation.

Simulation performance

The impact of an acute three-day CHO manipulation on performance in rugby has not been previously investigated during matches or in simulated matches. A strong trend towards failure to maintain performance during the second-half after the LCHO treatment was observed. This decrease in performance was 4.5% with an effect size of 0.7. This study was limited by a small subject numbers of ten (initially 12 were enrolled in the study), and also by the simulation instrument which proved to be less intense than match play (Deutsch, 2000; Deutsch, et al., 1996; McLean, 1992). Therefore, this result may be of practical importance to performance outcomes during a match. Most coaches or players seeking optimal performance throughout the entirety of a match would be interested in any strategy that could potentially prevent a 4.5% decrease in physical performance during the second half of a match.

Other MSS, however, have received more research attention. Ice hockey performance has been observed to improve following a CHO-enriched diet, containing $\sim 8 \text{ g.kg}^{-1}$, compared to a mixed diet of $\sim 6 \text{ g.kg}^{-1}$

(Åkermærk, et al., 1996). Soccer has also received research focus, early observations found that players who began a match with a low initial muscle (vastus lateralis) glycogen concentration covered 25% less distance compared to those beginning exercise with greater initial muscle glycogen concentration. In particular, a more marked difference was noted for running speed. Players with low glycogen content covered half the total distance walking, and 15% at maximal speed compared with 27% walking, and 24% sprinting for the players with high initial glycogen levels (Saltin, 1973). Also, performance in a small-sided (4-a-side) soccer match has also been found to be positively influenced by a high CHO dietary regimen that elevates pre-game muscle glycogen concentration (Balsom, et al., 1999). Performance, in this instance, was determined by the amount of time spent performing high intensity activities. Although this represents a relatively small proportion (~10%) of the total playing time, the importance of this type of exercise is clearly evident when one considers that nearly all scoring opportunities are created in conjunction with a period of high intensity exercise. The authors suggested that the availability of glycogen in type II fibres might have become limiting and impaired the ability to perform high intensity exercise. Unfortunately, post-game muscle biopsies were not taken during this study; therefore, the proposed mechanism involved in fatigue remains speculative.

Evidence is now beginning to accumulate supporting the hypothesis that elevating pre-exercise muscle glycogen may delay the onset of fatigue in MSS. The controlled environment of the laboratory has also provided evidence that time to fatigue during repeated bouts of short duration high intensity exercise is influenced by pre-exercise muscle glycogen concentration (Bangsbo, et al., 1992b; Jenkins, et al., 1993; Nicholas, et al., 1997).

It is important, however, to keep the current study in context. Forward play in rugby involves repeated voluntary muscular contractions with a multitude of intensities and variations. Furthermore, voluntary contraction is a complex series of events, and fatigue can occur at various sites in the pathway from the brain, to the motor endplate and within the muscle, ultimately affecting force generation. In this study, we have attempted to manipulate muscle glycogen, yet this only represents one treatment capable of delaying fatigue.

The unfortunate destruction of the mounted muscle samples - intended for fibre type specific glycogen analysis - prevented a more detailed examination of the specific cause of the observed decrease in performance in this study. Research involving a range of intensities and W:R ratios suggests that these variables, and in particular the intensity of exercise, have a major effect on the pattern of depletion among different fibre types. However, it appears from cycling experiments that an intermittent exercise pattern may actually be glycogen sparing compared to a continuous exercise pattern (Essen, 1978). This is most likely due to the availability of other substrates such as, glucose, fatty acids, triglycerides, and creatine phosphate within the muscle cell. Although both Type I, and II fibres are recruited during intense exercise this study suggests that Type II fibres may have a greater involvement in the initial tension development required to rapidly generate momentum during intermittent activity. This area of research undoubtedly warrants further investigation.

The simulation was designed to impose the physiological demands of a match, and also to provide the opportunity for subjects to intermittently perform supra-maximal movements which in turn were measured, and factored to give a performance score (TWS). A major limitation of such a protocol is in the validity of the performance

measure: how well do changes in an isolated rugby specific physical task, performed during a simulation, translate into on-field performance? The simulation used in his study slightly under-represented the gross physical demands of rugby. In addition, many of the technical aspects of forward play were impossible to replicate in a simulation. Therefore, the strong trend for maintenance of performance during the latter part of the simulation whilst on the HCHO treatment may have proved statistically significant if the true demands of rugby were represented in the simulation

The trend for maintenance of performance for 80 min of a rugby simulation that was observed in this study following the HCHO dietary treatment, as compared to the LCHO treatment, now awaits confirmation by subsequent investigations. If confirmed, then the mechanism(s) involved will require deeper investigation.

6 Conclusion

The present study demonstrates that a three-day HCHO dietary regimen significantly elevates muscle glycogen concentration above that of a LCHO dietary treatment in male rugby forwards. Subsequent performance in a simulated rugby match demonstrated that when subjects began exercise with a higher muscle glycogen concentration, performance was sustained throughout the entire 80 minutes. The implication of these results is that dietary intake in the days leading up to a rugby match could be crucial in attaining peak performance for forwards.

This study provides the basis for further research investigating the influence acute dietary regimens can have on performance in rugby. Future avenues of study could include determination of glycogen utilisation during a match, including specific fibre type glycogen utilisation patterns; investigation of the capacity of players who suffer damaged muscles from impacts, to resynthesise muscle glycogen before the next match; and comparison of nutritional interventions to optimise recovery from a match. The results also suggest that future studies should record, and report dietary practices in the days prior to testing the efficacy of acute nutritional supplementation - such as CHO supplementation during exercise.

To conclude, our understanding of the physiological and nutritional demands of rugby is, at best, limited. A comprehensive research effort combining laboratory, and field based designs is necessary to assist players and coaches in their quest for optimal performance.

References

- Åkermark, C., Jacobs, I., Rasmusson, M., Karlsson, J. (1996) Diet and muscle glycogen concentration in relation to physical performance in Swedish elite ice hockey players. *International Journal of Sport Nutrition* 6(3): 272-84.
- Balsom, P.D., Seger, J.Y., Sjodin, B., Ekblom, B. (1992) Physiological responses to maximal intensity intermittent exercise. *European Journal of Applied Physiology & Occupational Physiology* 65(2): 144-9.
- Balsom, P.D., Wood, K., Olsson, P., Ekblom, B. (1999) Carbohydrate intake and multiple sprint sports: with special reference to football (soccer). *International Journal of Sports Medicine* 20(1): 48-52.
- Bangsbo, J., Graham, T.E., Kiens, B., Saltin, B. (1992a) Elevated muscle glycogen and anaerobic energy production during exhaustive exercise in man. *Journal of Physiology* 451: 205-27.
- Bangsbo, J., Norregaard, L., Thorsoe, F. (1992b) The effect of carbohydrate diet on intermittent exercise performance. *International Journal of Sports Medicine* 13(2): 152-7.
- Bell, W. (1995) The estimation of body density in rugby union football players. *British Journal of Sports Medicine* 29(1): 46-51.
- Bell, W., Cobner, D., Cooper, S.-M. Anaerobic performance and body composition of international rugby union players. In: Reilly T, Clarys J, Stibbe A, eds. Science and football II. London: E. & F. N. Spon, 1993: 15-20.
- Bergstrom, J. (1962) Muscle electrolytes in man. *Scandinavian Journal of Clinical Laboratory Investigation* 14(suppl. 16): 1-110.
- Bergstrom, J., Hermansen, L., Hultman, E., Saltin, B. (1967) Diet, muscle glycogen and physical performance. *Acta Physiologica Scandinavica* 71(2): 140-50.

- Bergstrom, J., Hultman, E. (1966) Muscle glycogen synthesis after exercise: an enhancing factor localized to the muscle cells in man. *Nature* 210(33): 309-10.
- Bergstrom, J., Hultman, E., Roch-Norland, A.E. (1972) Muscle glycogen synthase in normal subjects: basal values, effect of glycogen depletion by exercise and of a carbohydrate-rich diet following exercise. *Scandinavian Journal of Clinical Laboratory Investigation* 29: 231-6.
- Bogdanis, G.C., Nevill, M.E., Boobis, L.H., Lakomy, H.K., Nevill, A.M. (1995) Recovery of power output and muscle metabolites following 30 s of maximal sprint cycling in man. *Journal of Physiology* 482(Pt 2): 467-80.
- Bosch, A.N., Dennis, S.C., Noakes, T.D. (1994) Influence of carbohydrate ingestion on fuel substrate turnover and oxidation during prolonged exercise. *Journal of Applied Physiology* 76(6): 2364-72.
- Burke, L.M., Gollan, R.A., Read, R.S. (1991) Dietary intakes and food use of groups of elite Australian male athletes. *International Journal of Sport Nutrition* 1(4): 378-94.
- Carlson, B.R., Carter, J.E.L., Patterson, P., Petti, K., Orfanos, S.M., Noffal, G.J. (1994) Physique and motor performance characteristics of US national rugby players. *Journal of Sport Sciences* 12: 403-412.
- Casagrande, G., Viviani, F. (1993) Somatotype of Italian rugby players. *The Journal of Sports Medicine and Physical Fitness* 33: 65-69.
- Cheetham, M.E., Williams, C. (1987) High intensity training and treadmill sprint performance. *British Journal of Sports Medicine* 21(2): 14-7.
- Christensen, E.H., Hansen, O. (1939) Arbeitsfahigkeit und Errichtung. *Skandinavische Archiv fur Physiologie* 8: 160-171.
- Costill, D.L., Miller, J.M. (1980) Nutrition for endurance sport: carbohydrate and fluid balance. *International journal of Sports Medicine*. 1: 2-14.
- Cowden, G. (2000) Changing face of rugby. *Sydney Morning Herald*.
- Deutsch, M.U. (2000) Time-motion analysis and physiological assessment of Super 12 and club rugby players. Newspaper.

Deutsch, M.U., Maw, G.J., Jenkins, D., Reaburn, P. (1996) Heart rate, blood lactate, and kinematic data of elite under 19 (Colts) match play. *Journal of Sport Sciences* 16: 561-570.

Docherty, D., Wenger, H.A., Neary, P. (1988) Time-motion analysis related to the physiological demands of Rugby. *Journal of Human Movement studies* 14: 269-277.

Essen, B. (1978) Glycogen depletion of different fibre types in human skeletal muscle during intermittent and continuous exercise. *Acta Physiologica Scandinavica* 103(4): 446-55.

Evans, W.J., Pinney, S.D., Young, V.R. (1982) Suction applied to a muscle biopsy maximises sample size. *Medicine and Science in Sports and Exercise* 14: 101-102.

Flynn, M.G., Costill, D.L., Hawley, J.A., Fink, W.J., Neufer, P.D., Fielding, R.A., Sleeper, M.D. (1987) Influence of selected carbohydrate drinks on cycling performance and glycogen use. *Medicine & Science in Sports & Exercise* 19(1): 37-40.

Frail, H. (1993) Dietary requirements for rugby players. *New Zealand Journal of Sports Medicine* 21(4): 58-60.

Friden, J., Seger, J., Ekblom, B. (1989) Topographical localization of muscle glycogen: An ultrahistochemical study in the human vastus lateralis. *Acta Physiologica Scandinavica* 135(3): 381-391.

Gibson, H., Edwards, R.H.T. (1985) Muscular exercise and fatigue. *Sports Medicine* 2: 120-132.

Green, H., Bishop, P. (1976) Time-motion and physiological assessments of ice hockey performance. *Journal of Applied Physiology* 40(2): 159-63.

Greenhaff, P.L., Ren, J., Söderlund, K., Hultman, E. (1991) Energy metabolism in single human muscle fibers during contraction without and with epinephrine infusion. *Am. J. Physiol.* 260(23): E713--E718.

Holmyard, D.J., Hazeldine, R.J. Seasonal variations in the anthropometric and physiological characteristics of international rugby union players. In: Reilly T, Clarys J, Stibbe A, eds. *Science and football II*. London: E. & F. N. Spon, 1993: 21-6.

Jardine, M.A., Wiggins, T.M., Myburgh, K.H., Noakes, T.D. (1988) Physiological characteristics of rugby players including muscle glycogen content and muscle fibre composition. *South African Medical Journal* 73(9): 529-32.

Jenkins, D.G., Palmer, J., Spillman, D. (1993) The influence of dietary carbohydrate on performance of supramaximal intermittent exercise. *European Journal of Applied Physiology & Occupational Physiology* 67(4): 309-14.

Kirkendall, D.T., Foster, C., Dean, J.A., Gorgan, J., Thompson, N.N. Effect of glucose polymer supplementation on performance of soccer players. In: Reilly R, eds. *Science and Football*. London: Spon, 1988:

Leatt, P.B., Jacobs, I. (1989) Effect of glucose polymer ingestion on glycogen depletion during a soccer match. *Canadian Journal of Sport Science* 14(2): 112-116.

Maud, P.J. (1983) Physiological and anthropometric parameters that describe a rugby union team. *British Journal of Sports Sciences* 17(1): 16-23.

Maughan, R.J. (1997) Energy and macronutrient intakes of professional football (soccer) players. *British Journal of Sports Medicine* 31(1): 45-7.

Maughan, R.J., Greenhaff, P.L., Leiper, J.B., Ball, D., Lambert, C.P., Gleeson, M. (1997) Diet composition and the performance of high-intensity exercise. *Journal of Sports Sciences* 15(3): 265-75.

Maughan, R.J., Poole, D.C. (1981) The effects of a glycogen loading regimen on the capacity to perform anaerobic exercise. *European Journal of Applied Physiology* 46: 211-219.

McLean, D.A. (1992) Analysis of the physical demands of international rugby union. *Journal of Sports Sciences* 10(3): 285-96.

Morton, A.R. (1978) Applying physiological principals to rugby training. *Sports Coach* 2(2): 4-9.

Muckle, D.S. (1973) Glucose syrup ingestion and team performance in soccer. *British Journal of Sports Medicine* 7: 340-343.

Nicholas, C.W., Green, P.A., Hawkins, R.D., Williams, C. (1997) Carbohydrate intake and recovery of intermittent running capacity. *International Journal of Sports Nutrition* 7: 251-260.

- Pizza, F.X., Flynn, M.G., Duscha, B.D., Holden, J., Kubitz, E.R. (1995) A carbohydrate loading regimen improves high intensity, short duration exercise performance. *International Journal of Sport Nutrition* 5(2): 110-6.
- Quarrie, K.L., Handcock, P., Toomey, M.J., Waller, A.E. (1996) The New Zealand rugby injury and performance project. IV. Anthropometric and physical performance comparisons between positional categories of senior A rugby players. *British Journal of Sports Medicine* 30(1): 53-6.
- Reid, R.M., Williams, R. (1974) A concept of fitness and it's measurement in relation to rugby football. *British Journal of Sports Medicine* 8: 96-99.
- Rigg, P., Reilly, T. A fitness profile and anthropometric analysis of first and second class rugby union players. In: Reilly T, Lees A, Davids K, eds. *Science and football*. London: E. & F. N. Spon, 1988: 194-200.
- Roberts, D. (1997) Nutrition knowledge, information sources, and dietary practices of New Zealand rugby football union academy players. Newspaper.
- Saltin, B. (1973) Metabolic fundamentals in exercise. *Medicine and Science in Sports and Exercise* 5: 137-45.
- Sherman, W.M., Costill, D.L., Fink, W.J., Hagerman, F.C., Armstrong, L.E., Murray, T.F. (1983) Effect of a 42.2 km footrace and subsequent rest or exercise on muscle glycogen and enzymes. *Journal of Applied Physiology* 55: 1219-1224.
- Sherman, W.M., Costill, D.L., Fink, W.J., Miller, J.M. (1981) Effect of exercise-diet manipulation on muscle glycogen and its subsequent use during performance. *International Journal of Sports Medicine* 2: 114-118.
- Treadwell, P.J. Computer-aided match analysis of selected ball games (soccer and rugby union). In: Reilly T, Lees A, Davids K, Murphy WJ, eds. *Science and Football*. London: E. and F. N. Spon, 1988: 282-287.
- van Erp-Baart, A.M.J., Saris, W.H.M., Binkhorst, R.A., al, e. (1989) Nationwide survey on nutritional habits in elite athletes: Part 1 Energy, carbohydrate, protein and fat intake. *International Journal of Sports Nutrition* 10(1): 3s-10s.
- Wilson, N.C., Russell, D.G., Wilson, B.D. 1993 Body size and shape of New Zealanders. Dunedin, NZ:

Appendices

Appendix A Participant Information sheets

Diet and Rugby Study information

Muscle biopsy information

Medical History Questionnaire

Diet and Rugby Study

We would like to invite you to take part in a study we are undertaking to investigate the role of diet in Rugby Union performance.

Correct conditioning is recognised as a crucial aspect in preparing for a competitive game of rugby union. However the role of the diet in Rugby performance is poorly understood. The purpose of the current investigation is to determine if a diet high in carbohydrate will influence performance in a rugby simulation (for locks and loose forwards) when compared to a diet that contains only moderate amounts of carbohydrate.

Each volunteer will be required to complete two 80 minute indoor rugby simulation trials. Each trial will be separated by a week and for the three days preceding each trial volunteers will be asked to consume (and record) a specific diet. Each volunteer will consume one diet before the first trial, then the alternative diet for the second trial. Some of the foods for the dietary treatments will be provided.

The simulation trial is designed to mimic the demands you face when playing a game of rugby therefore this simulation should act as ideal preseason training. Volunteers will be filmed by a video camera during the simulation trial in order to determine performance. Also blood lactate measurements will be undertaken during the simulation. This will involve taking a small drop of blood from a pin-prick on the earlobe, we expect to take 4-5 samples from each volunteer during the simulation. Due to the small number of pain receptors in the ear this is a painless procedure, and sterile equipment will be used for each sample.

Volunteers will also be required to undergo a muscle biopsy from the thigh muscle. The biopsies will be taken immediately before and immediately after each simulation trial. The biopsy involves first receiving an injection of local anaesthetic. A small cut is made (~10 mm). From this a small piece of muscle tissue (the size of a match head) is removed and the incision is closed with a piece of tape especially made to close small wounds. After the biopsy you will be able to carry out your normal daily activities, however bathing and swimming should be avoided. The area should be kept dry and it is recommended that the area be covered with plastic when showering. It is also recommended that you avoid heavy contact training for three days.

Before volunteering for the biopsy procedure, there are some things that we would like all volunteers to consider. Some cultures, societies, and/or religions have laws relating to the removal of tissue from the body. These laws may state that the unnecessary removal of tissue from the body is forbidden, or that certain restrictions are involved such as keeping the removed tissue for the duration of life. Other groups may also place restrictions on blood sampling. Please note that the experiment requires all volunteers to provide small blood samples. Individuals volunteering for the biopsy must understand that the tissue taken during the biopsy will be dissolved during the analysis and **cannot** be returned at the conclusion of the study. We can provide photographs etc. if this poses a desirable compromise. If you have **any** questions relating to these issues, please discuss them with the investigators before volunteering.

All volunteers will be required to perform a VO₂ max test in the laboratory. This will take approximately 40 minutes. The VO₂ max test will involve running on a treadmill for approximately 10-15 minutes. The speed of the treadmill will begin at a slow jog, and increase in speed every 2 minutes until you are fatigued and choose to end the test. During this test a gas analyser will be used to measure the amounts of oxygen and carbon dioxide in the air you breathe out. To ensure the safety of all participants and experimenters, all subjects taking part will be asked questions regarding diabetes, AIDS, hepatitis etc.

Each volunteer will receive \$150 travel/time monetary compensation for the lab tests, muscle biopsy, and for the simulation trial. Post study you will also be offered the opportunity to attend a free sports nutrition seminar/workshop. If at any time you want to withdraw from the study you are free to do so. If you wish, copies of your results will be given to you as they come available. A copy of the report on the study will also be made available.

This study has received approval, from the SRHA (OTAGO) Ethics Committee. Please feel free to contact the researcher if you have any questions about this study.

Principal Researcher:

Mr. Glenn Kearney
Dept. of Human Nutrition
University of Otago
PO Box 56
DUNEDIN
Ph (Wk): 479 8358
Ph (Hm): 477 3420

Supervisor:

Dr. Nancy Rehrer
School of Physical Education
University of Otago
55 Union Court
DUNEDIN
Ph (Wk): 479 9128

Dr Christine Thomson
Dept. of Human Nutrition
University of Otago
PO Box 56
DUNEDIN

Ph (Wk): 479 7943

Muscle biopsy information

The analytical technique described in the protocol for muscle glycogen concentrations and fibre types require molecular biological techniques which can only be done on tissue sections or homogenates. Muscle tissue from the vastus lateralis shall be obtained before the simulation and immediately after with a biopsy needle (5 mm) according to Evans et al. (1982).

The muscle biopsy technique as described here has been used by Dr H. Keizer routinely in research (eg. patients with myopathies and diabetes), as well as with healthy volunteers, before and after exercising (Keizer et al., 1987; Brouns et al., 1989). In most instances these were longitudinal studies in which subjects had up to four biopsies per experiment and 18 per research project. The risk for the patient is very limited; this is supported by the fact that many elite athletes also undergo this procedure. This procedure has also been previously used before and after rugby matches (Jardine et al., 1988) and before and after competitive Ice Hockey matches (Åkermark et al., 1996).

Dr David Gerrard has performed at least 50 muscle biopsies during the last 12 months with no infections or complications.

References

Brouns F, Saris WHM, Beckers E et al. Metabolic changes induced by sustained exhaustive cycling and diet manipulation. *Int. J. Sports Med.* 1989; 10 (s1): s49-62.

Evans WM, Pinney SK, Young VR. Suction applied to a muscle biopsy maximises sample size. *Med Sci Sports Exerc.* 1982; 14: 101-102.

Keizer H, Kuipers H, Van Kranenburg G, Geurten P. Influence of liquid and solid meals on muscle glycogen resynthesis, plasma fuel hormone response and maximal physical working capacity. *Int J Sports Med.* 1987; 8: 99-104.

Jardine MA, Wiggins TM, Myburgh KH, Noakes TD. Physiological characteristics of rugby players including muscle glycogen content and muscle fibre composition. *South African Med J.* 1988; 73(9): 529-532.

Åkermark C, Jacobs I, Rasmusson M, Karlsson J. Diet and muscle glycogen concentration in relation to physical performance in Swedish elite ice hockey players. *I J Sports Nutr* (1996); 6: 272-284.

Medical History Questionnaire

The purpose of this questionnaire is to ensure the safety of all participants and experimenters. The results of this questionnaire are strictly confidential, and will be destroyed immediately following subject recruitment for the study.

Please answer the following questions carefully and honestly.

Name: _____

Phone number: _____

Date of Birth: _____

Please answer the following questions as accurately as possible. If the answer to any of these questions is yes, please state whether the condition is current or past, the length of time you have had the condition, and any treatment that you may be receiving for that condition.

Have you ever had, or do you currently have:

Hepatitis (a, b, or c)	Yes	/	No

Jaundice	Yes	/	No

HIV or AIDS	Yes	/	No

Glandular fever (If within last 6 months)	Yes	/	No

Diabetes	Yes / No

Other infectious conditions (Please specify)	Yes	/	No

_____ Thank you for your cooperation,
Glenn Kearney

Appendix B Consent Form

Participant Consent Form

I _____ have read and understand the information sheet for volunteers participating in the experiment: **The Effect of diet on Rugby Performance**. I have had the chance to ask questions and they have been answered to my satisfaction.

I understand that participation is voluntary and that I may withdraw from the experiment at any time and this will in no way effect my academic progress nor sport related opportunities offered by the School of Physical Education or Otago Rugby.

I understand that my participation in this study is confidential and that no material which could identify me as a subject will be used in any reports on this study.

I understand that this investigation will be stopped should it appear that it is harmful to me.

"I _____ (full name) hereby consent to take part in this study."

Signed _____ Date _____

Researchers: Mr Glenn Kearney, Dr Nancy Rehrer, Dr Christine Thomson,
Dr David Gerrard

Contact phone numbers:

Mr Kearney: (03) 479 8358

Dr Rehrer: (03) 479 9128

Dr Thomson: (03) 479 7943

Dr Gerrard (03) 479 8938

Appendix C Diet Record booklet

INSTRUCTIONS FOR KEEPING A 4 DAY DIET RECORD

I.D.....

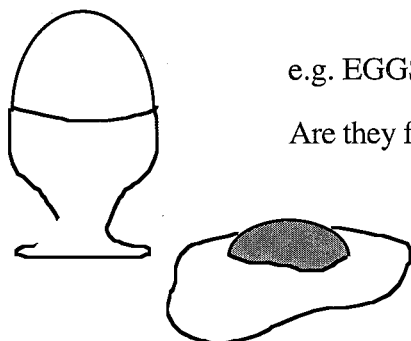
RECORD SHEET

PLEASE READ THESE IMPORTANT INSTRUCTIONS CAREFULLY

- * Please record ALL food and drinks consumed
- * Please record the food at the time of eating and NOT from memory at the end of the day
- * You should include all meals & snacks, plus sweets, drinks (including water) etc.
- * Remember to include any additions to foods already recorded such as: sauces, dressings or extras e.g. gravy, salad dressings, stuffings, sugar, honey, syrups etc., butter or margarine (e.g. added to bread, crackers, vegetables).
- * If you do not eat a particular meal or snack, simply draw a line across the page at this point. This will show that you definitely have not eaten anything.

DESCRIBING FOOD AND DRINK – GUIDELINES

1. Please give details of the method of cooking all foods (e.g. fried, grilled, boiled, roasted, steamed, poached, stewed).
2. Give as many details as possible about the **type** of food that you eat e.g. brand name of food where applicable (e.g. Miracle margarine);
type of: Breakfast cereal (e.g. Weetbix)
milk (e.g. whole milk or 'trim milk')
cake or biscuit (e.g. fruit cake, wheatmeal biscuit)
fruit (e.g. fresh, canned, dried, stewed)
soft drink (e.g. regular or low calorie)
3. Name the type of cheese, fish or meat (e.g. cheddar, cod fillet, loin of pork)



e.g. EGGS

Are they fried, boiled, poached or scrambled?

RECORDING THE AMOUNTS OF FOODS YOU EAT

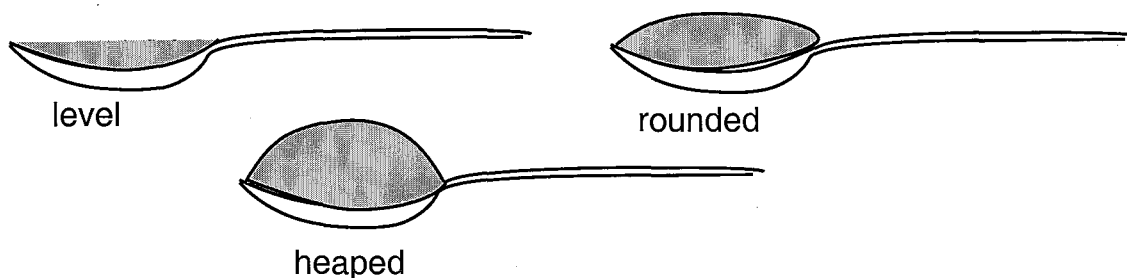
It is also very important to record the quantity of each food and drink you consume.

Here are some suggestions on how to record amounts:

- **IN HOUSEHOLD MEASUREMENTS**

For many foods such as vegetables, cereals and canned or stewed fruit, a household measurement is adequate.

e.g. STATE THE NUMBER OF TEASPOONS (t), TABLESPOONS (T), CUPS etc. State whether spoons are level, rounded or heaped.



Butter and margarine can be measured in teaspoons or tablespoons if you find this an easy method.

- **WEIGHTS MARKED ON PACKAGES**

All convenience foods have their weight marked on the packaging and this can be quoted e.g. half a 425g can of baked beans.

- **BREAD** - indicate the size of the slices (e.g. sandwich, medium, toaster).
- **CHEESE, MEAT & FISH**

If at all possible, it would be very helpful to weigh your portions of these foods.

If this is not possible, please **use the pictures on the attached sheets** to indicate what sort of portion sizes you eat e.g. you might have 1 portion of spaghetti size A, 1 portion of meat size B or 2 slices of cheese size C.

- **USE COMPARISONS** for describing portion sizes where this is easier e.g. potato - size of a hen's egg, cheese - size of a matchbox.

IT IS VERY IMPORTANT THAT YOU DO NOT ADJUST WHAT YOU EAT AND DRINK BECAUSE YOU ARE KEEPING A RECORD. THIS IS VERY EASY TO DO, BUT REMEMBER, WE ARE INTERESTED IN YOUR EATING HABITS, NOT THE PERFECT DIET!!!

DAY 1 - Date

- ° Record ALL food and drink consumed during the day including sweets, snacks, 'nibbles', sauces and dressings.
- ° Please record: METHOD OF COOKING (e.g. *boiled* pasta)
 TYPE OF FOOD (e.g. *boiled wholegrain* pasta)
 QUANTITY OF FOOD (e.g. *1 cup* boiled wholegrain pasta)

MEAL/ SNACK	QUANTITY EATEN	DETAILS OF FOOD AND DRINK
EARLY MORNING		
BREAKFAST		
DURING MORNING		
LUNCH		

Appendix D Treatment diets

Test diet 1 - Food list & instructions

Test diet 2 - Food list & instructions

Test diet 1

Test diet 2

Test diet 1 - Food list & instructions

Name..... Wt.....kg

Daily amounts:-these are **minimum** amounts- see list of extra foods below.

** Trim milk.....	mls
** Bread	slices
** Raw fruit	pieces
** Jam, honey or syrup.....	tablespoons
** Dried fruit	half cup
** Sugar.....	tablespoons

Important:

On Training Diet 1 it is important to eat the foods listed with ** in (at least) the amounts given. If you are feeling hungry on this diet then eat more of these foods: -

Cereal, Rice, pasta

Starchy vegs - kumara, yams, corn

Fruit - any sort, either raw, tinned, dried, or stewed.

Bread, scones, pikelet, loaf

Yoghurt

Jam, honey or syrup

Sugar, boiled lollies, fruit leathers, popcorn

NB Avoid the consumption of **alcohol** over this three day period. See instructions on plastic bag for your breakfast on the morning of the simulation. The sports drink is ideal straight after any training session you undertake.

Test diet 2 - Food list & instructions

Name.....

Wt.....kg

Daily amounts:

Standard milk..... mls

Bread slices

Raw fruit pieces

Potatoes..... average

Ice cream..... 1 1/2 scoops in one ice cream cone.

Important:

On Training Diet 2 it is important to eat the foods listed with ## in ONLY the amounts given and no more. If you are feeling hungry on this diet then eat more of the other foods see list below:-

Meat, including ham & other cold meats, Chicken

Fish, including sardines, tuna, salmon

Cheese, Eggs

Lettuce, tomatoes, green peppers, other salad vegetables.

Mayonnaise, peanut butter (no added sugar), peanuts

Cream, Butter or margarine

NB. Do **not** add extra **sugar** to your drinks and **avoid** the consumption of **alcohol** over this three day period. See instructions on plastic bag for your breakfast on the morning of the simulation.

Test diet 1

Days 1, 2 & 3: Foods marked ** & in **bold** are to be eaten freely.

Breakfast

- ** Large serving **cereal** (.....cups)
- ** Tinned **fruit** (.....cup)
- ** **Milk**
- ** **Toast**
- Butter / margarine
- ** **Jam / honey**
- Tea / coffee / milo / water / fruit juice

Snack

- ** **Sports drink**(.....ml)
- ** **Fruit**

Lunch

- ** Large serving **pasta** (.....cups, cooked)
- Ready made sauce
- Small serving grated cheese (no more than 4 tablespoons)
- Green salad, tomatoes
- ** **Raw fruit**
- ** **Bread**
- Butter / margarine
- ** **Jam / honey**
- Tea / coffee / milo / water / fruit juice

Snack

- ** **Sports drink** (.....ml)
- Yoghurt or ** **dried fruit**

Dinner

- Average serving of meat, chicken or fish - (should take up no more than a quarter of your plate)
- ** Large serving **Rice** (.....cups, cooked)
- Ready made sauce
- Serving green vegetables
- ** Large serving of **starchy vegetables** (especially kumara, yams, parsnip, peas or corn)
- ** **Ice cream** (.....cups)
- ** **Tinned fruit** (.....cup)
- Tea / coffee / milo / water / fruit juice

Snack

- ** **Sports drink** (.....ml)
- ** **Toast or bread, muffin, pikelet, crumpet, loaf.** Butter / margarine
- ** **Jam / honey & Popcorn / **fruit**

Test diet 2

Days 1, 2 & 3: Foods marked **##** are restricted, those in **bold** may be eaten freely.

Breakfast

##Weetbix only

##Milk / **Cream**

Egg, boiled, poached or scrambled

##Toast (bread from amount on list)

Butter / margarine

Peanut butter or vegemite

Tea / coffee / milo / water / tomato juice

Snack

Tea / coffee / milo / water / tomato juice

##Fruit from amount on list

Lunch: Day 1

1 only Thin crust pizza with extra topping -**cheese, ham, salami, tomato** (no pineapple) & lunch extra's.

Lunch: Day 2

2 only Bread rolls -with **chicken, ham, meat, mayonnaise, lettuce, tomato**, etc. & lunch extra's.

Lunch: Day 3

##1 can Chilly Beans and 2 tacos -with grated **cheese & sour cream, lettuce, tomato** etc. & lunch extra's.

Lunch extra's: Days 1-3

Extra green salad, tomatoes

##Raw fruit from

list

##Bread from list

Butter/ margarine/ peanut butter/ Vegemite.

Tea/Coffee/Milo/Water/Tomato juice

Snack

Tea / coffee / milo / water / tomato juice

Cheese

Dinner

Large serving of **meat, chicken or fish**

##Potato -quantity from list with margarine or butter

Large serving **green vegetables** (no peas or broad beans)

##Serving of root vegetables (no kumara, yams, parsnip or corn)

##Ice cream 1¹/₂ scoops in a cone

Tea / coffee / milo / water / tomato juice

Snack

Tea/ coffee/ water/ tomato juice/ milo made with milk or **cream**

Cheese/ sardines/ salmon/ tuna/ tomatoes on toast

##**Butter / margarine**

Appendix E Individual TWS results

Individual TWS (total weighted score) results

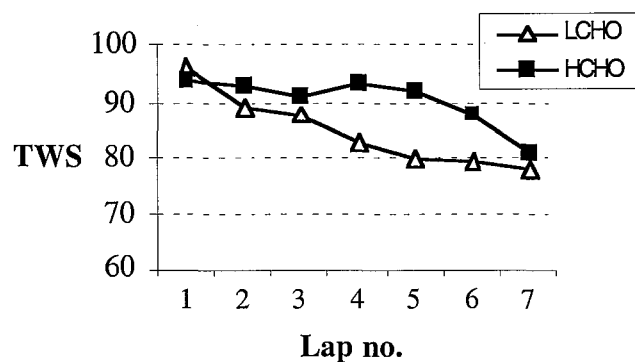


Figure Y1 Subject 1

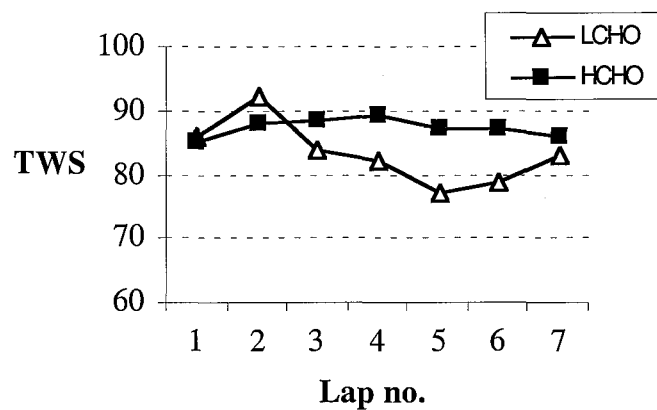


Figure Y2 Subject 2

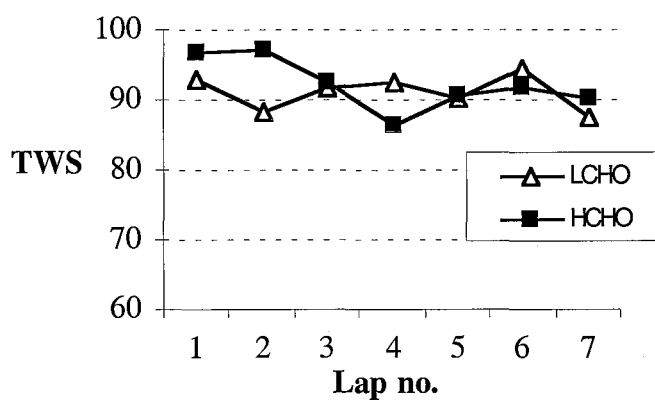


Figure Y3 Subject 3

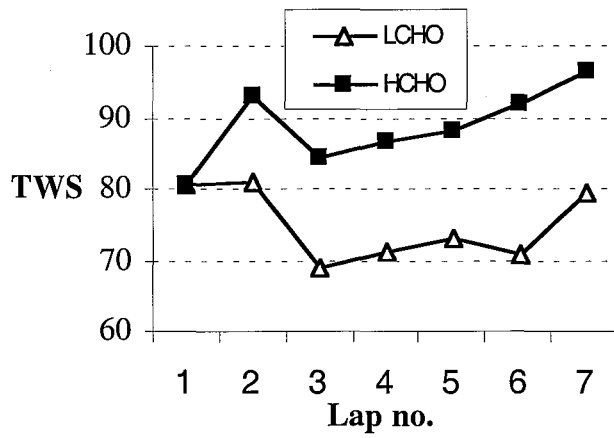


Figure Y4 Subject 4

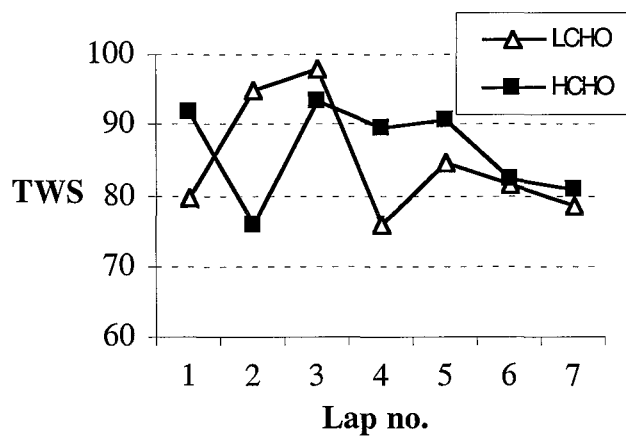


Figure Y5 Subject 5

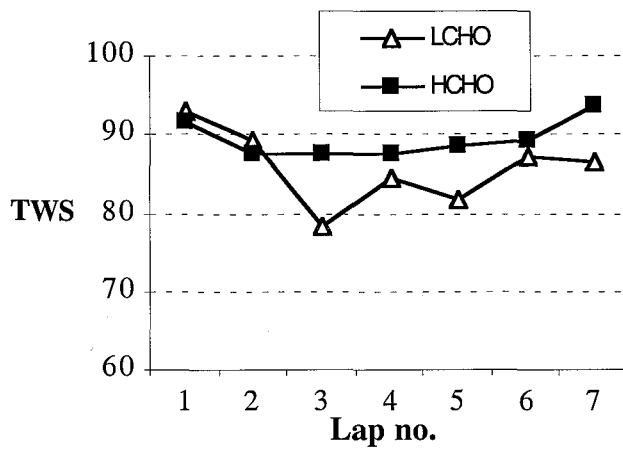


Figure Y6 Subject 6

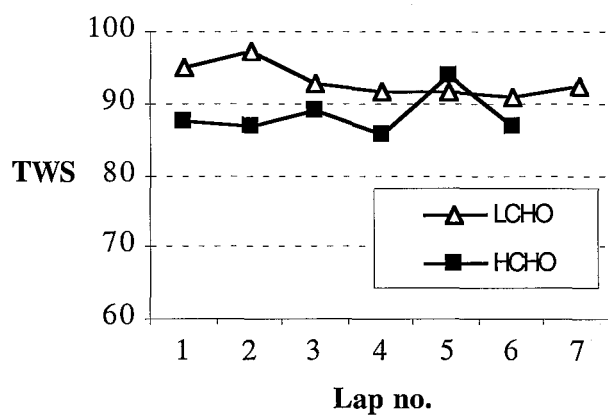


Figure Y7 Subject 7

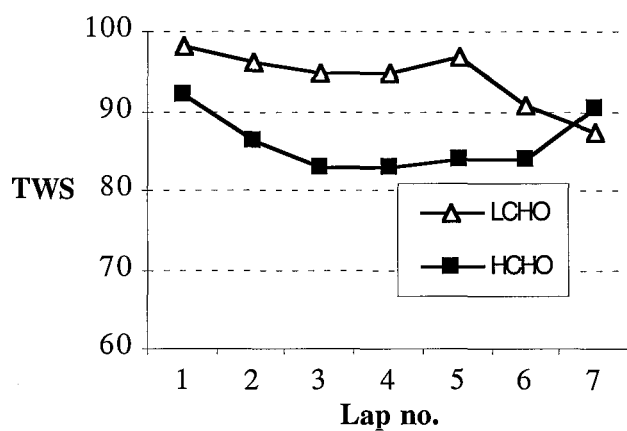


Figure Y8 Subject 8

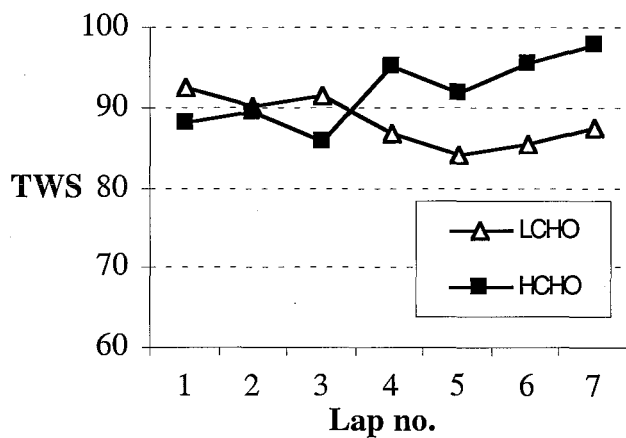


Figure Y9 Subject 9

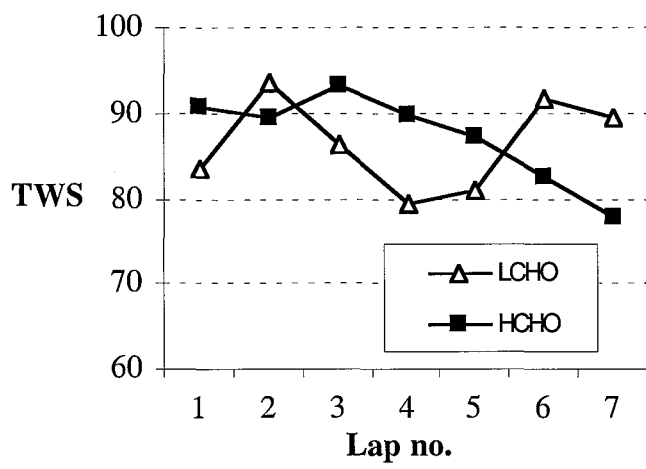


Figure Y10 Subject 10